SCREENING-LEVEL HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT FOR THE PASSAIC RIVER STUDY AREA

VOLUME I
DRAFT REPORT

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Volume I

TABLE OF CONTENTS (CONT'D)

TABLE OF CONTENTS (CONT'D)

TABLE OF CONTENTS (CONT'D)

LIST OF FIGURES

LIST OF TABLES

LIST OF TABLES (CONT'D)

LIST OF TABLES (CONT'D)

List of Acronyms

ACOE	Army Corps of Engineers
ADI	average daily intakes
AET	apparent effects threshold
AHH	aryl hydrocarbon hydroxylase
AOC	Administrative Order on Consent
ARAR	applicable, relevant, and appropriate requirements
BAF	bioaccumulation factor
BAZ	biologically active zone
BCF	bioconcentration factor
BSAF	biota sediment accumulation factor
CAE	chemical assimilation efficiency
CBR	critical body residues
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CNS	central nervous system
CPC	chemicals of potential concern
CSF	cancer slope factor
CSO	combined sewer outfalls
DO	dissolved oxygen
ECAO	Environmental Criteria and Assessment Office
EPA	United States Environmental Protection Agency
EqP	equilibrium partitioning
ER-L	effect range-low
ER-M	effect range-median
ERA	ecological risk assessment
GC-MS	Gas Chromapography/Mass Specprometry
HEAST	Health Effects Assessment Summary Tables
HERA	Human Health and Ecological Risk Assessment
HHAG	Human Health Assessment Group
HHRA	human health risk assessment
HI	hazard index
HQ	hazard quotient

LIST OF ACRONYMS (CONT'D)

I-TEF	International-toxicity equivalency factor
IRIS	Integrated Risk Information Service
ISC	Interstate Sanitation Commission
IWP	Investigation Work Plan
LADI	lifetime average daily intakes
LMS	linearized multistage
LOAEL	lowest-observed-adverse-effect-level
MS/MSD	Matrix Spike/Matrix Spike Duplicate
MS/D	Matrix Spike and Laboratory Duplicate
NAS	National Academy of Sciences
NCEA	National Center for Environmental Assessment
NCP	National Contingency Plan
NJDEP	New Jersey Department of Environmental Protection
NOAA	National Oceanic and Atmospheric Administration
NOAEL	no-observed-adverse-effect-level
OCC	Occidental Chemical Corporation
PAS	Princeton Aqua Science
POTW	publicly owned treatment works
QA	quality assurance
QC	quality control
QSAR	quantitative structure activity relationships
RCRA	Resource Conservation and Recovery Act
RfD	reference doses
RME	reasonable maximum exposures
SAV	submerged aquatic vegetation
SOW	Statement of Work
SQG	sediment quality guidelines
STORET	Storage and Retrieval of Water Quality Data
TEF	toxicity equivalency factor
UCL	upper confidence limit
USGS	United States Geological Survey

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1.0 INTRODUCTION

This document contains a screening-level Human Health and Ecological Risk Assessment (HERA) for the Passaic River Study Area, herein referred to as the Site. This screening-level HERA was prepared by ChemRisk® pursuant to the Administrative Order on Consent (AOC), dated April 20th, 1994, between Environmental Protection Agency (EPA) and Occidental Chemical Corporation (OCC). Consistent with the goals of the AOC and Statement of Work (SOW), the objectives of the screening-level HERA are to characterize the potential risks to human and ecological receptors potentially exposed to chemicals present in sediments, water, and aquatic organism at the Site. In addition to the characterization of potential risks, the results of the screening-level HERA will provide the risk management decision-makers for the Site with several key pieces of information, including the following:

- evaluation of the number and types of chemicals as well as other physical and chemical stressors, and their relative contribution to the overall risk to human and ecological receptors;

- identification of those human populations and ecological receptors for which the potential risks may be greatest, based on their behavior, location, and potential for exposure;

- identification of the media and exposure pathways that contribute the greatest to potential human health and/or ecological risks; and,

- evaluation of the potential risks from a myriad of chemicals that are attributable to a number of ongoing municipal and industrial sources.

An evaluation of available data regarding exposure media (i.e., water, sediment, and biota), and concentrations of chemicals within the exposure media is presented in Section 2.0 (Data Compilation and Evaluation). Quantitative and qualitative estimates of potential risks to human health and ecological receptors are presented in Sections 3.0 (Screening-Level Human Health Risk Assessment) and Section 4.0 (Screening-Level Ecological Risk Assessment), respectively, based

on the potential uptake of chemicals from sediment and water through the food web by key aquatic organisms, and from consumption of fish and invertebrates by humans. By way of background and information, a discussion of the Site history and setting are provided below.

1.1 Site Setting

The Site (Figure 1-1) is located on the lower portion of the Passaic River, one of the tributaries of Newark Bay, in the Greater New York City Metropolitan Area. The Site is defined as that portion of the Passaic River extending from the abandoned ConRail Bridge (located approximately 4,000 feet upriver from the red channel junction marker at the confluence of the Hackensack and Passaic Rivers) to a transect six miles (31,680 feet) upriver of this bridge. The Site is situated within five navigation reaches, defined by the Army Corps of Engineers (ACOE), including Point No Point Reach, Harrison Reach, Newark Reach, Kearny Reach, and Arlington Reach. The Site is considered navigable by ACOE (1987).

The Passaic River drains a 935 square mile watershed encompassing 117 municipalities in eight counties in northeastern New Jersey, and 15 municipalities and two counties in southern New York. Based on data from the United States Geological Survey (USGS) (1989) and ACOE (1987), the upstream Passaic River contributes the majority of freshwater inflow (approximately 1,200 cubic feet per second on average) to the tidal (lower) portion of the River, which includes the Site. Additional freshwater inflow comes from tributaries located downstream of the Dundee Dam, including the Third River, the Second River, Franks Creek, and Lawyers Creek, and from urban runoff, including storm sewers and combined sewer outfalls (CSOs).

Land use along the lower Passaic River, extending south of the Dundee Dam and including the Site, is dominated by high-density commercial and industrial/commercial development, as depicted in Photographs 1 through 10. There is little or no public access to the River. The left bank of the Site (looking upstream), much of which was once primarily marshland, is almost fully developed (ERM, 1992). Active or abandoned industrial properties and rail lines completely dominate the majority of the right bank (looking upstream) of the Site. A highly developed network of highways, CSOs, stormwater outfalls, and publicly owned treatment works (POTWs) exists throughout the area (Mueller et al., 1982).

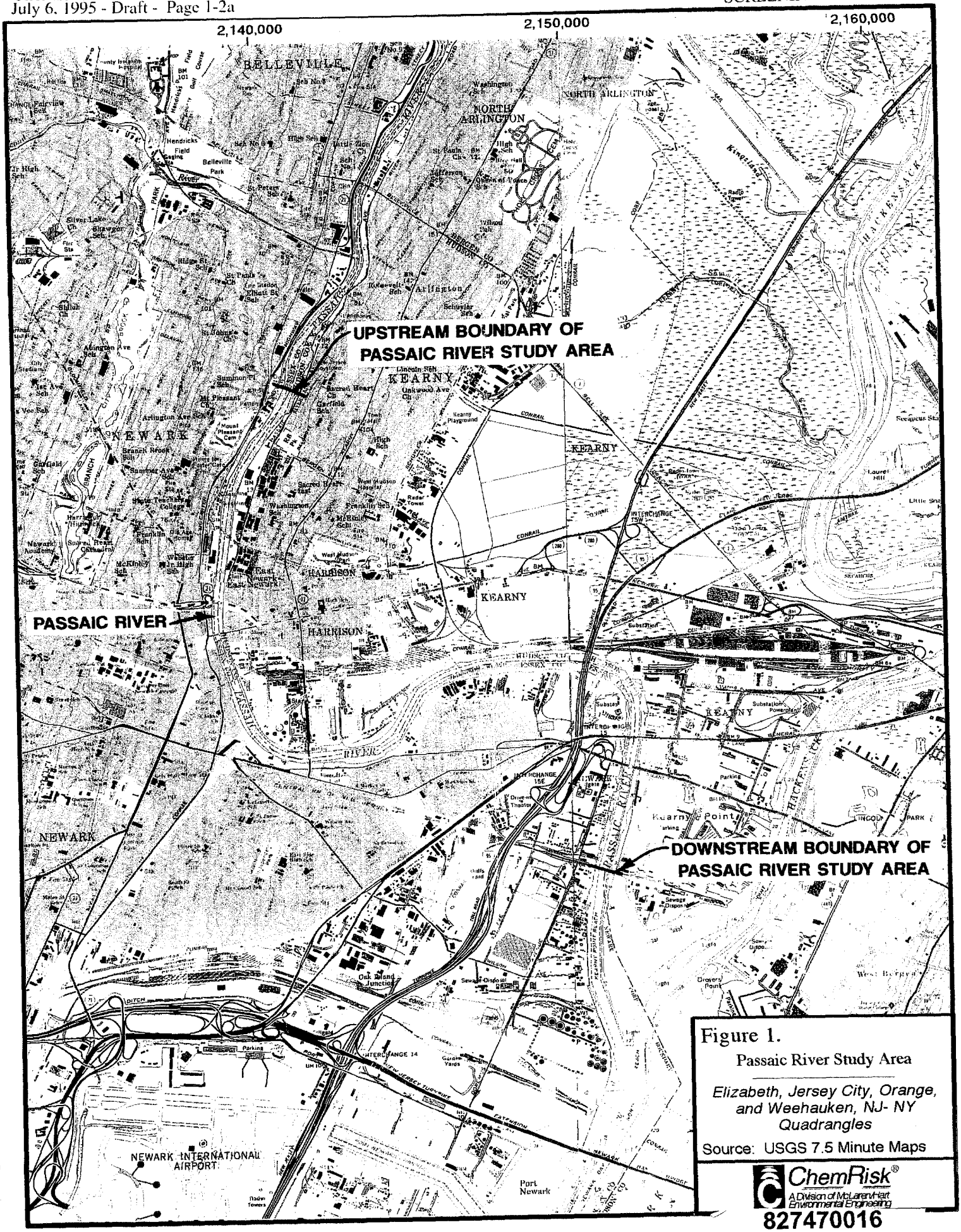

Figure 1.

Passaic River Study Area

Elizabeth, Jersey City, Orange, and Weehauken, NJ- NY Quadrangles

Source: USGS 7.5 Minute Maps

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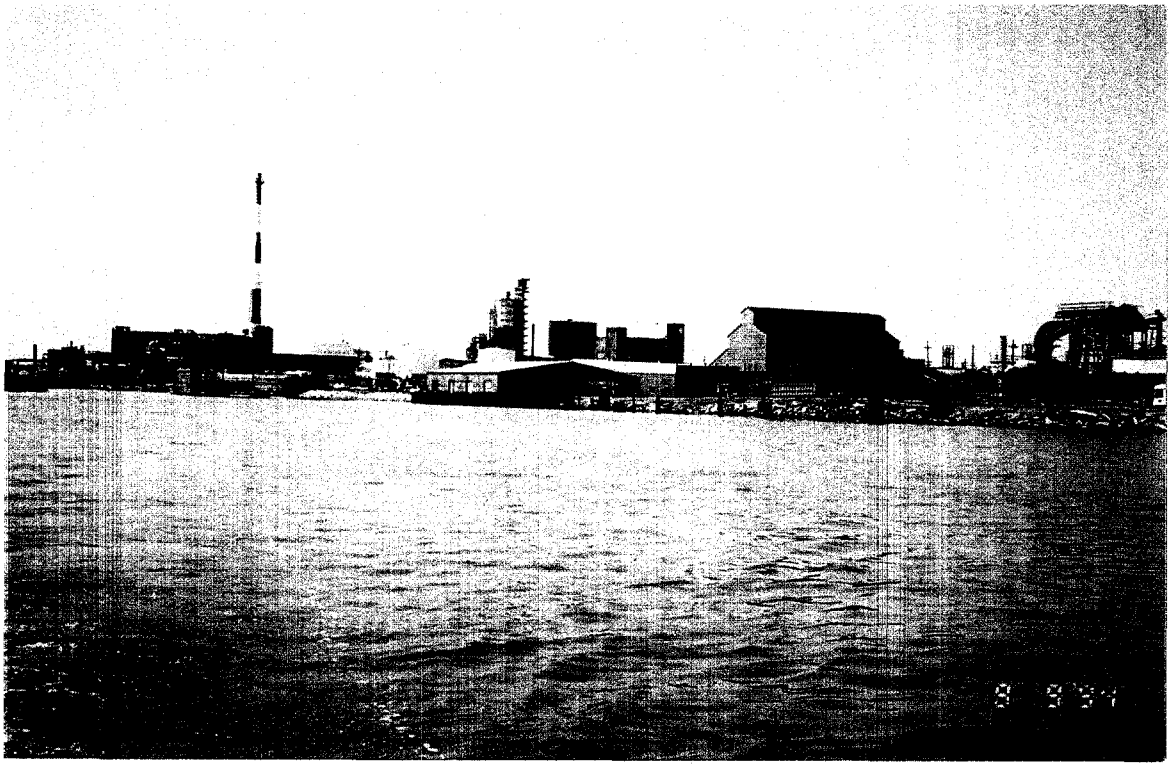

Photo #1: Passaic River Study Area--Point No Point Reach
Beginning of the Passaic River Study Area--Left Bank

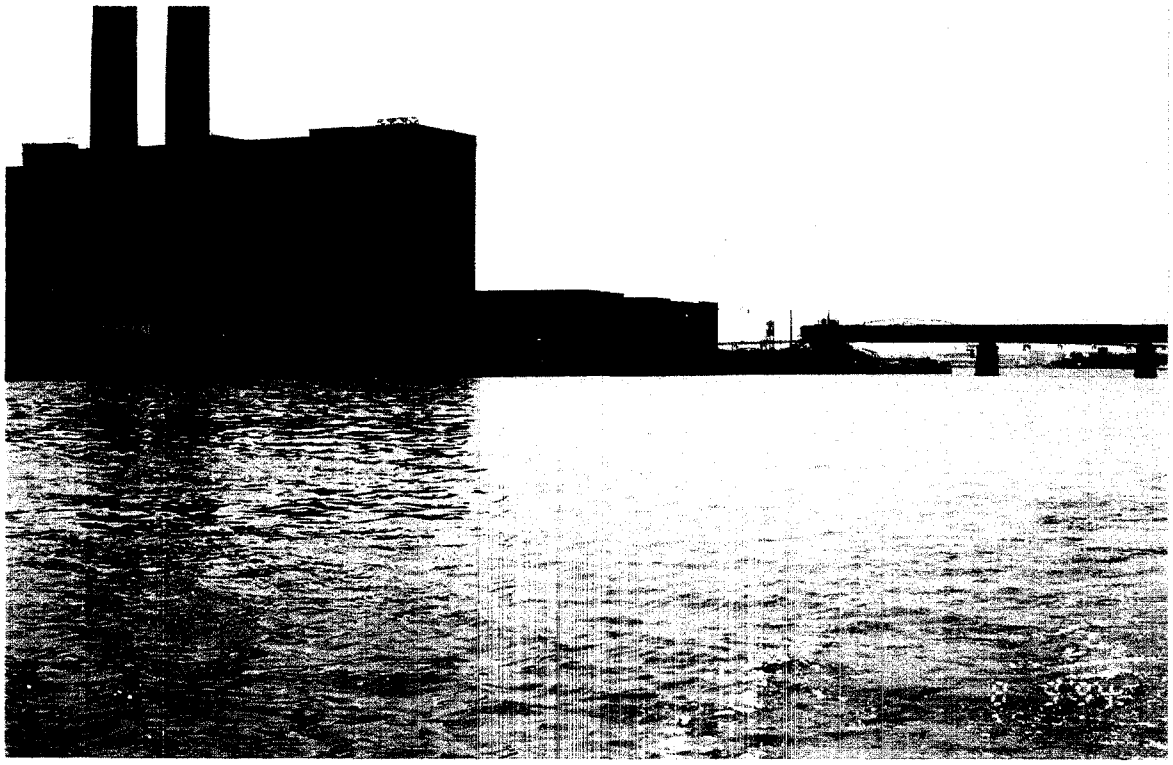

Photo #2: Passaic River Study Area--Point No Point Reach
Beginning of the Passaic River Study Area--Right Bank

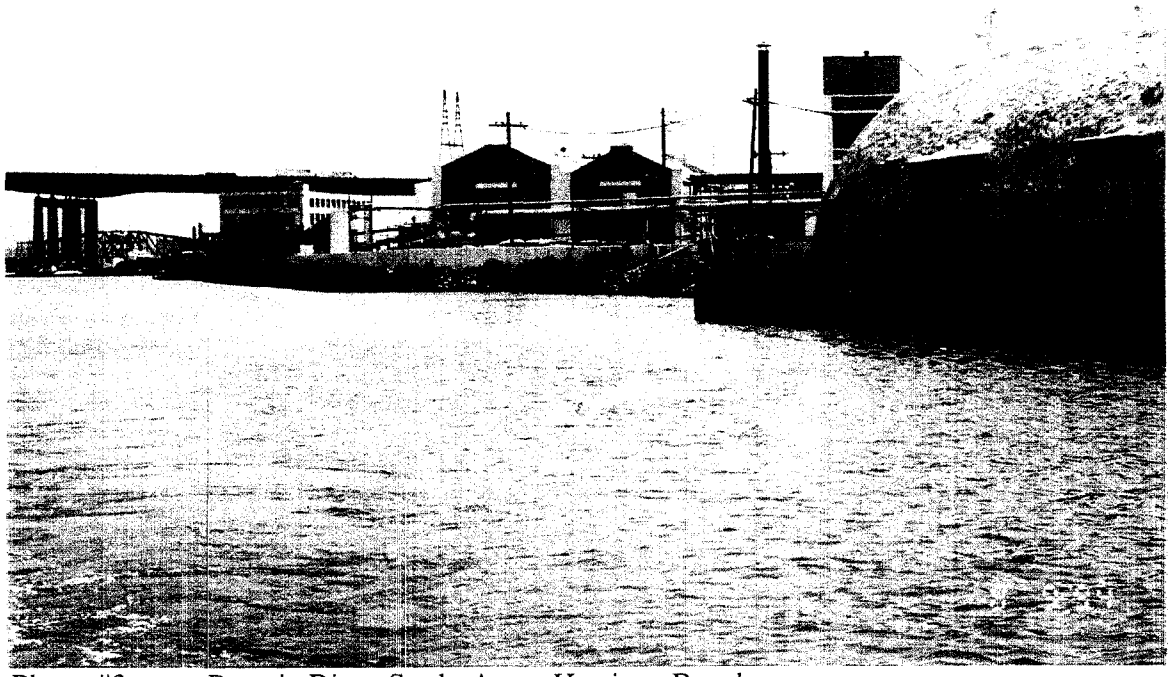

Photo #3: Passaic River Study Area--Harrison Reach
At Mile 2--Left Bank

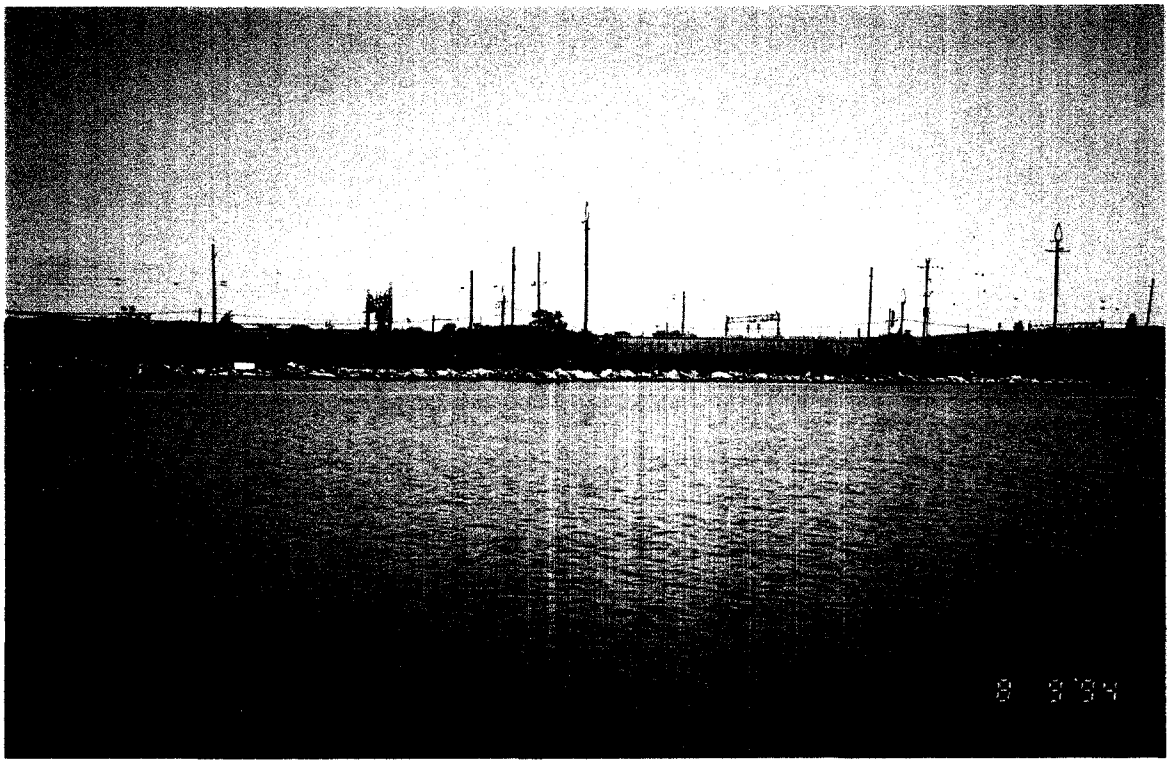

Photo #4: Passaic River Study Area--Harrison Reach
At Mile 2--Right Bank

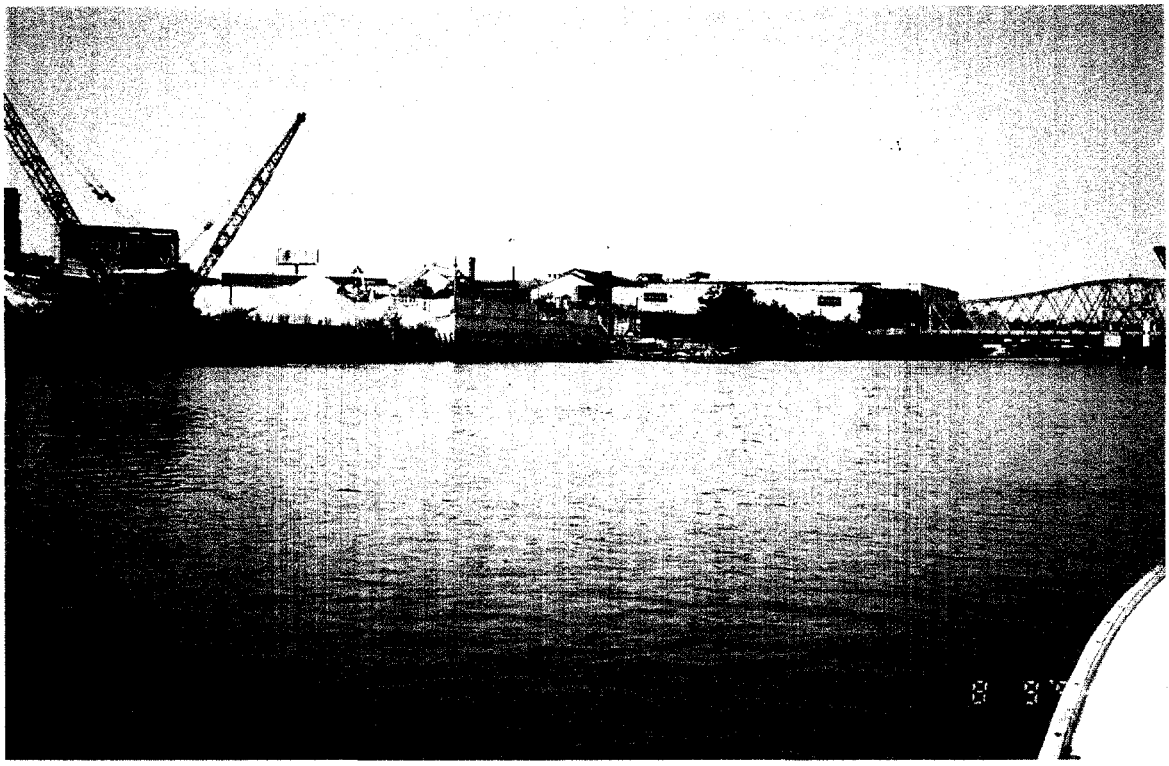

Photo #5: Passaic River Study Area--Newark Reach
Between the William Stickel Memorial Bridge and the Clay St. Bridge--Left Bank

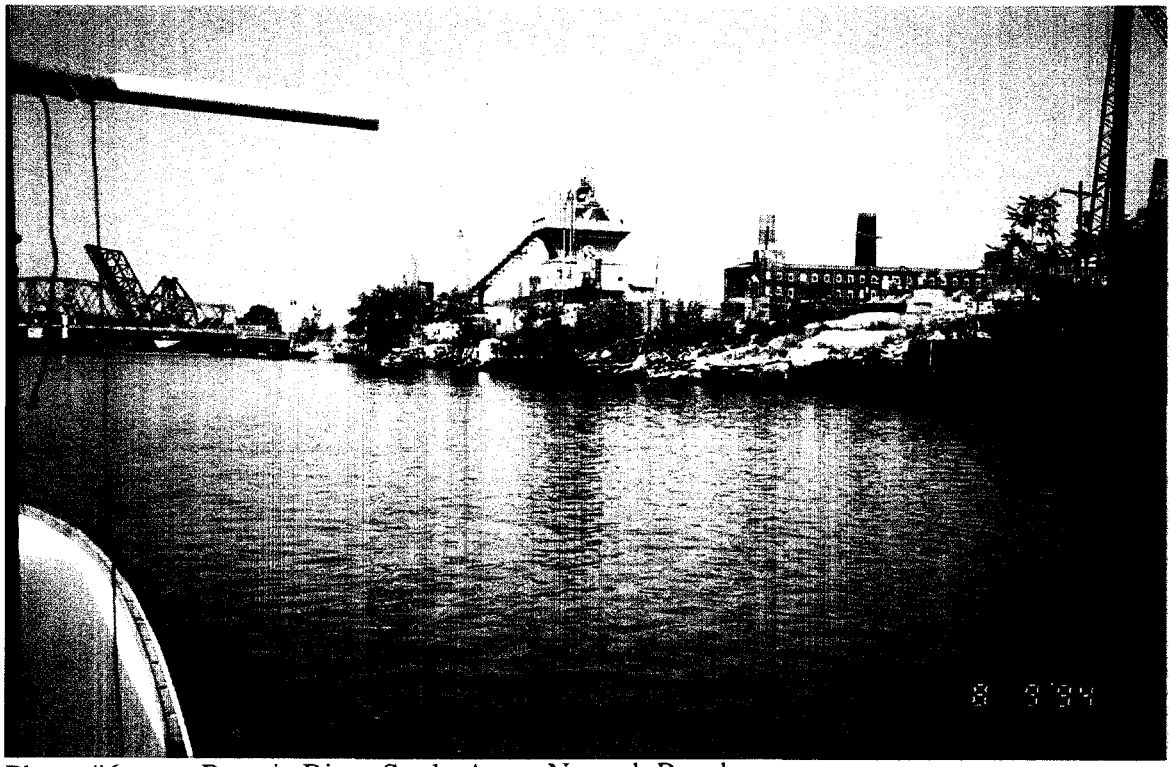

Photo #6: Passaic River Study Area--Newark Reach
Between the William Stickel Memorial Bridge and the Clay St. Bridge--Right Bank

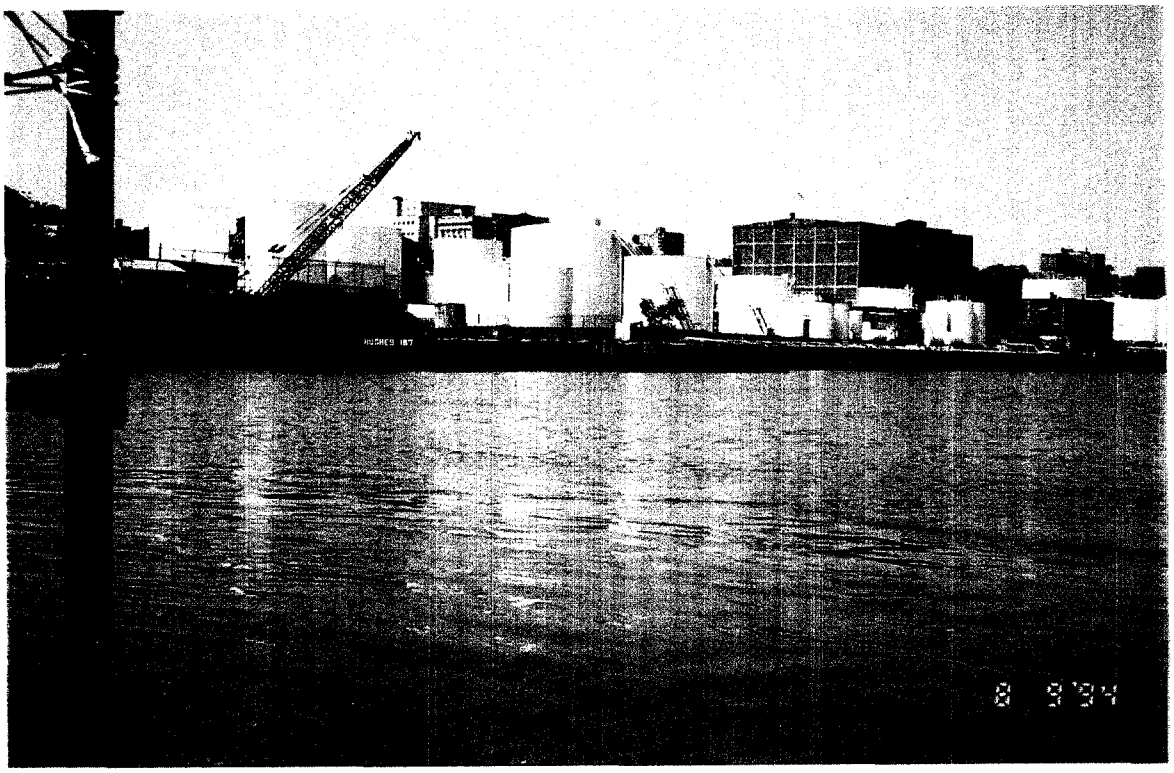

Photo #7: Passaic River Study Area--Kearny Reach
Upstream from the Erie & Lackawanna Railroad Bridge--Left Bank

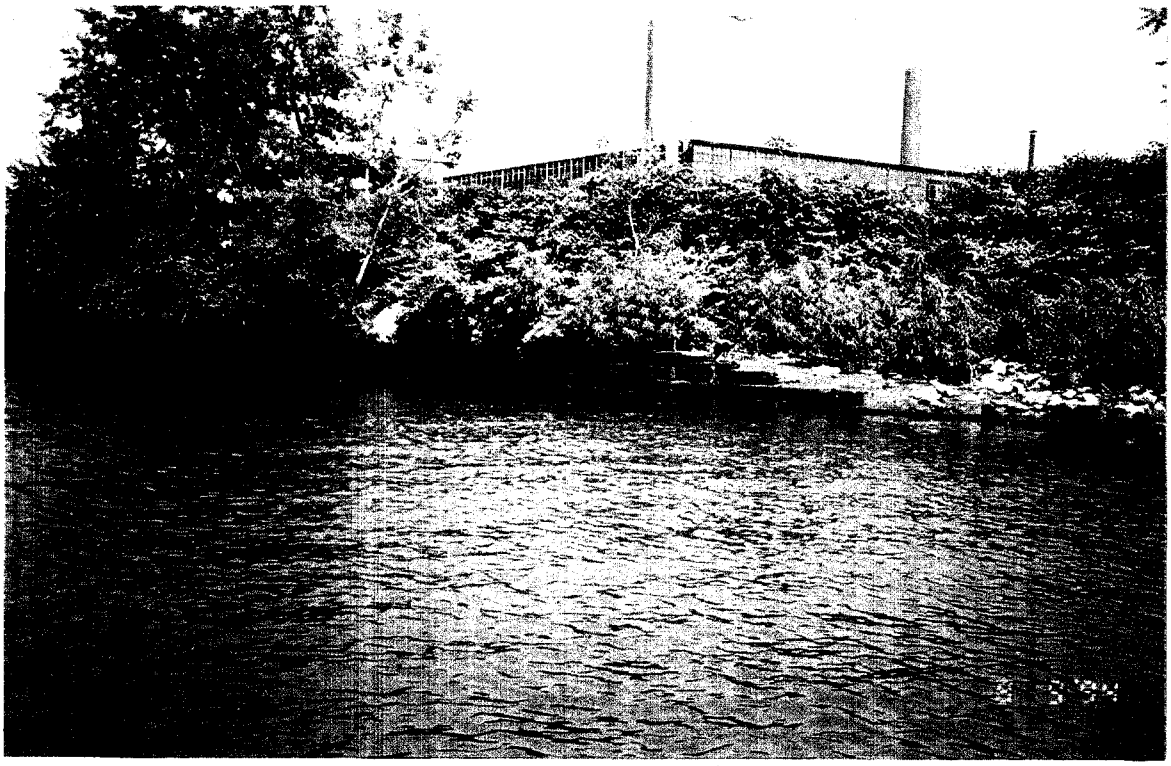

Photo #8: Passaic River Study Area--Kearny Reach
Upstream from the Erie & Lackawanna Railroad Bridge--Right Bank

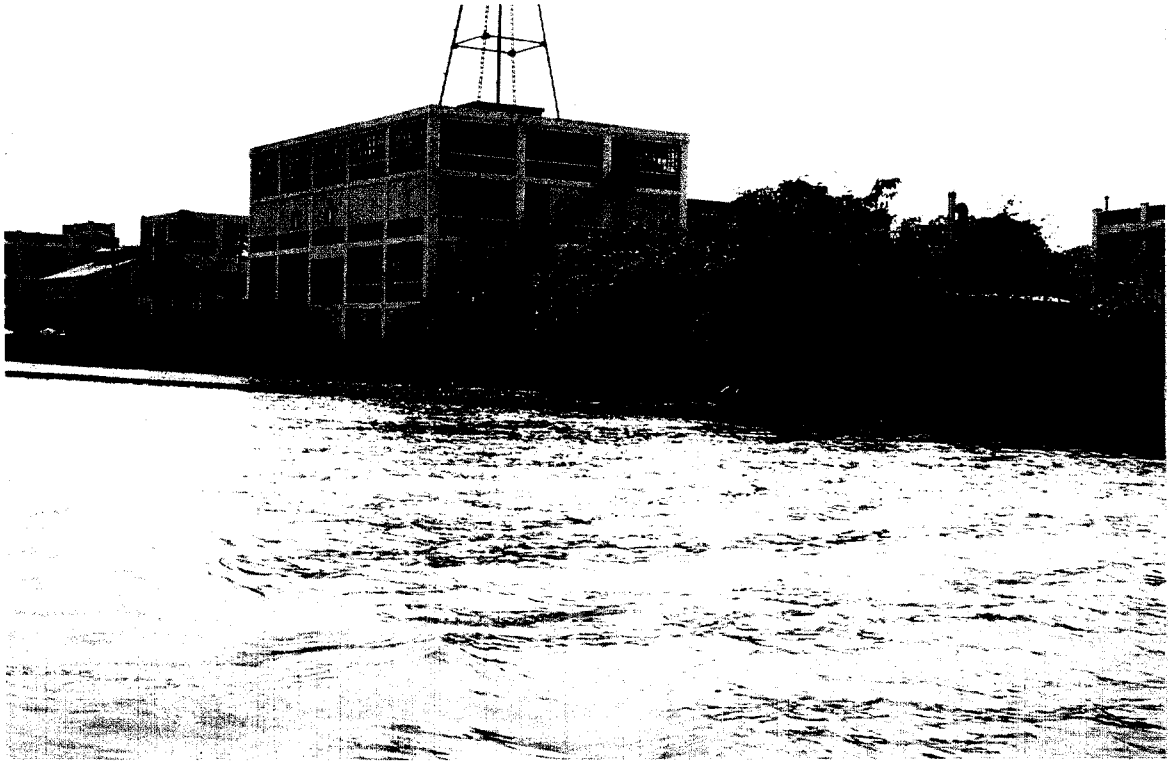

Photo #9: Passaic River Study Area--Arlington Reach
End of Passaic River Study Area--Left Bank

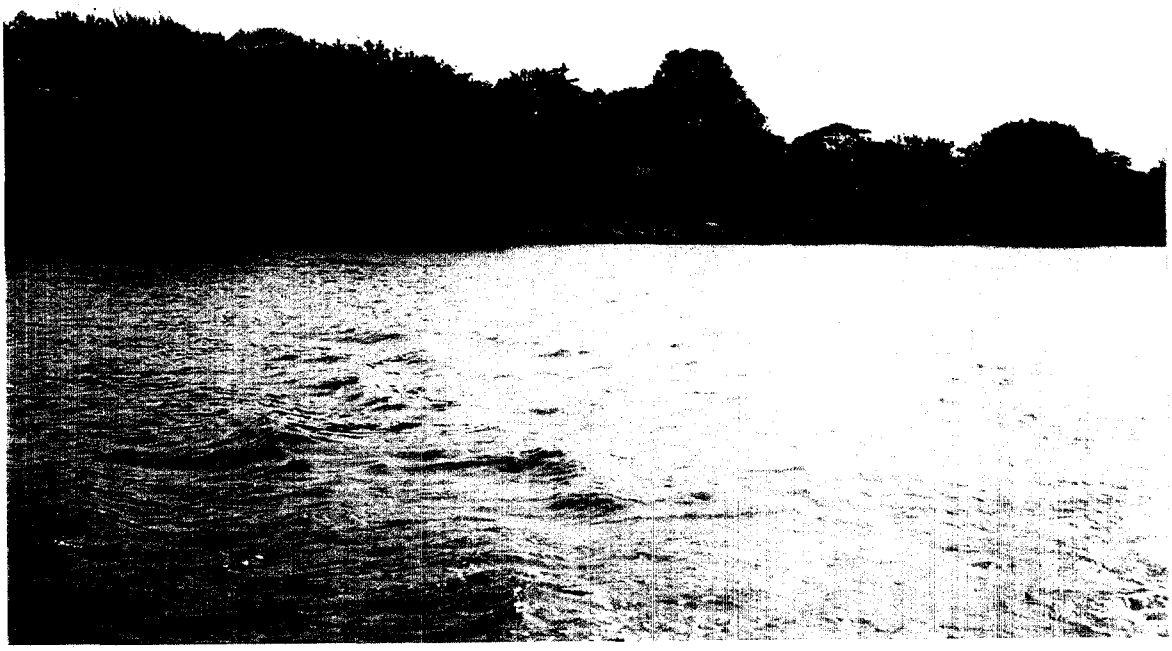

Photo #10: Passaic River Study Area--Arlington Reach
End of Passaic River Study Area--Right Bank

The Site has a long history of industrialization, dating back more than two centuries (Meyers, 1945; Cunningham, 1966a, 1966b; Brydon, 1974; Crawford et al., 1995). By the turn of the century, Newark was the largest industrial-based city in the United States with well established industries such as petroleum refining, shipping, tanneries, creosote wood preservers, metal recyclers, and manufacturing of materials such as rubber, rope, textiles, paints and dyes, pharmaceutical, raw chemicals, leather, and paper products (Meyers, 1945; Cunningham, 1954; Cunningham, 1966a; Brydon, 1974; Halle, 1984; MacRae's, 1986; Galishoff, 1988). Both World War I and World War II promoted further urban and industrial growth in the region (Squires, 1981). Despite the development of sewage treatment plants, many industrial facilities located along the Passaic River were not connected to the Passaic Valley Sewage Commission trunk line until the late 1950s (Brydon, 1974). In addition, Newark's growing prominence as an industrial center was associated with a rapidly expanding population, resulting in the generation of increasing volumes of human wastes (Suszkowski et al., 1990).

As a result of historical industrial and urban growth, the lower Passaic River, including the Site, is considered to have serious water quality problems (ACOE, 1987). The water quality is rated very poor in both the freshwater regime above the Dundee Dam, and below the dam in the saline tidal reach (ACOE, 1987). Depressed levels of dissolved oxygen have been known to be a chronic problem in Newark Bay and its tributaries since the early 1900s (McCormick et al., 1983). In addition, as is true of numerous industrialized waterways in the United States, sediments within the Site contain elevated concentrations of numerous hazardous chemicals including, but not limited to, cadmium, copper, lead, mercury, nickel, zinc, bis(2-ethylhexyl)phthalate, polycyclic aromatic hydrocarbons, polychlorinated biphenyls (PCBs), 4,4´-dichlorodiphenyltrichloroethane (4,4´-DDT), diesel range organics (Total Extractable Petroleum Hydrocarbons), polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (PCDD/Fs), and chlorinated herbicides and phenols (Bonnevie et al., 1992; Bonnevie et al., 1993; Gillis et al., 1993; Huntley et al., 1993; Wenning et al., 1993; Bonnevie et al., 1994; Wenning et al., 1994; Gillis et al., 1995; Huntley et al., 1995; Iannuzzi et al., 1995).

In addition to degraded sediment and water quality, the expansion of industry and population surrounding the Site has resulted in a severe reduction in the availability of natural habitats for indigenous and migratory biota (Squires and Barclay, 1990; Crawford et al., 1994). As discussed in Section 4.0, almost all of the wetlands in the lower Passaic River have been eliminated, with

more than 7,500 acres developed since 1940 (ACOE, 1987). A decline in bird diversity in the area is attributed to the destruction of marshlands and other natural habitats as a result of encroachment of human development and industrial activities on nesting and breeding grounds (Burger et al., 1993). In addition, populations of fish and shellfish in the Site and surrounding area have been substantially reduced by over-harvesting, loss of habitat, and pollution (Mytelka et al., 1981; Esser, 1982; Franz, 1982).

In summary, the quality of all environmental media within and around the Site has been severely degraded over the past century or more due to industrialization. The adverse impacts have been caused by numerous chemical and physical stressors that cannot be related to a single facility or group of facilities. Therefore, any assessment of theoretical "health risks" associated with a given chemical at the Site must be presented in context of the more relevant and complex issue of the total quality of the Site and the reasons for its current state of degradation.

1.2 References for Section 1.0

ACOE. 1987. *Passaic River Basin, New Jersey and New York Phase I-General Design. Memorandum: Flood Protection Feasibility, Main Stem Passaic River, Main Report and Environmental Impact Statement.* U.S. Army Corps. of Engineers, New York District, NY. December.

Bonnevie, N.L., D.G. Gunster, and R.J. Wenning. 1992. Distribution of lead in surficial sediments from the Newark Bay estuary. *Environ. Internal.* 18:497-508.

Bonnevie, N.L., R.J. Wenning, S.L. Huntley, and H. Bedbury. 1993. Distribution of inorganic compounds in sediments from three waterways in Northern New Jersey. *Bull. Environ. Contam.Toxicol.* 51:672-680.

Bonnevie, N.L., S.L. Huntley, B.W. Found, and R.J. Wenning. 1994. Trace metal contamination in surficial sediments from Newark Bay, New Jersey. *Sci. Total Environ.* 144:1-16.

Brydon, N.F. 1974. *The Passaic River Past, Present, Future.* New Brunswick, NJ: Rutgers University Press.

Burger, J., K. Parsons, and M. Gochfeld. 1993. Avian populations and environmental degradation in an urban river: The kills of New York and New Jersey. In: *Avian Conservation.* J. Jackson (ed.). Madison, WI: University Wisconsin Press.

Crawford, D.W., N.L. Bonnevie, C.A. Gillis, and R.J. Wenning. 1994. Historical changes in the ecological health of the Newark Bay Estuary, New Jersey. *Ecotoxicol. Environ. Saf.* 29:276-303.
Crawford, D.W., N.L. Bonnevie, and R.J. Wenning. 1995. Sources of pollution and sediment contamination in Newark Bay, New Jersey. *Ecotoxicol. Environ. Saf.* 30:85-100.

Cunningham, J.T. 1954. *Made in New Jersey: The Industrial Story of a State.* New Brunswick, NJ: Rutgers University Press.

Cunningham, J.T. 1966a. *New Jersey: America's Main Road.* New York, NY: Doubleday & Company.

Cunningham, J.T. 1966b. *Newark.* Newark, NJ: New Jersey Historical Society.

ERM. 1992. *Hilton-Davis ECRA Report.* Environmental Resource Management Group. Prepared for the New Jersey Department of Environmental Protection and Energy, Trenton, NJ.

Esser, S.C. 1982. Long-term changes in some finfishes of the Hudson-Raritan estuary. In: *Ecological Stress and the New York Bight: Science and Management.* G.F. Mayer (ed.). Rockville, MD: National Oceanic and Atmospheric Administration.

Franz, D.R. 1982. A historical perspective on mollusks in Lower New York Harbor, with emphasis on oysters In: *Ecological Stress and the New York Bight: Science and Management* G. F. Mayer (ed.). Rockville, MD: National Oceanic and Atmospheric Administration.

Galishoff, S. 1988. *Newark: The Nation's Unhealthiest City, 1832-1895.* New Brunswick, NJ: Rutgers University Press.

Gillis, C.A., N.L. Bonnevie, S.H. Su, J.G. Ducey, S.L. Huntley, and R.J. Wenning. 1995. DDT, DDD, and DDE contamination of sediment in the Newark Bay Estuary, New Jersey. *Arch. Environ. Contam. Toxicol.* 28:85-92.

Gillis, C.A., N.L. Bonnevie, and R.J. Wenning. 1993. Mercury contamination in the Newark Bay Estuary. *Ecotoxicol. Environ. Saf.* 25: 214-226.

Halle, D. 1984. *America's Working Man: Work, Home, and Politics among Blue-Collar Property Owners.* Chicago, IL: University of Chicago Press.

Huntley, S.L., N.L. Bonnevie, H. Bedbury, and R.J. Wenning. 1993. Distribution of polycyclic aromatic hydrocarbons (PAHs) in three northern New Jersey waterways. *Bull. of Environ. Contam. and Toxicol.* 51:865-872.

Huntley, S.L., N.L. Bonnevie, and R.J. Wenning. 1995. Polycyclic aromatic hydrocarbon and petroleum hydrocarbon contamination in sediment from the Newark Bay Estuary, New Jersey. *Arch. Environ. Contam. Toxicol.* 28:93-107.

Iannuzzi, T.J., S.L. Huntley, N.L. Bonnevie, B.L. Finley, and R.J. Wenning. 1995. Distribution and possible sources of polychlorinated biphenyls in dated sediments from the Newark Bay Estuary, New Jersey. *Arch. Environ. Contam. Toxicol.* 28:108-117.

MacRae's. 1986. *MacRae's State Industrial Directory: New Jersey*. New York, NY: MacRae's Blue Book, Inc.
McCormick, J.M, Hires, R.I., Luther, G.W., and S.L. Cheng. 1983. Partial recovery of Newark Bay, NJ, following pollution abatement. *Mar. Poll. Bull.* 14(5):188-197.

Meyers, W.S. 1945. *The Story of New Jersey. Vols.1-4.* New York, NY: Lewis Historical Publishing.

Mueller, J.A., T.A. Gerrish, and M.C. Casey. 1982. *Contaminant Inputs to the Hudson-Raritan Estuary.* National Oceanic and Atmospheric Administration, Rockville, MD. Report No. OMPA-21. August.

Mytelka, A.I., M. Wendell, P.L. Sattler, and H. Golub. 1981. *Water Quality of the Hudson-Raritan Estuary.* National Oceanic and Atmospheric Administration. Boulder, CO. NOAA Grant #NA80RAD00034.

Squires, D.F. 1981. *The Bight of the Big Apple.* The New York Sea Grant Institute of the State University of New York and Cornell University, NY.

Squires, D.F. and J.S. Barclay. 1990. *Nearshore Wildlife Habitats and Populations in the New York/New Jersey Harbor Estuary.* Marine Science Institution and Department of Natural Resources Management and Engineering, The University of Connecticut, Storrs, CT. November.

Suszkowski, D.J., S. Cairns, and D. Heimbach. 1990. Conditions in New York-New Jersey Harbor Estuary. In: *Cleaning Up Our Coastal Waters: An Unfinished Agenda.* A Regional Conference, March 12-14, Manhattan College, Riverdale, NY.

USGS. 1989. *Water Resources Data New Jersey Water Year 1987: Volume I: Atlantic Slope Basins, Hudson River to Cape May.* U.S. Geological Survey, Water Resources Division, Reston, VA. USGS/WRD/HD-89-274. June.

Wenning, R.J., M.A. Harris, B.Finley, D.J. Paustenbach, and H. Bedbury. 1993. Application of pattern recognition techniques to evaluate polychlorinated dibenzo-p-dioxin and dibenzofuran distributions in surficial sediments from the Lower Passaic River and Newark Bay. *Ecotoxicol. Environ. Saf.* 25:103-125.

Wenning, R.J., N.L. Bonnevie, and S.L. Huntley. 1994. Accumulation of metals, polychlorinated biphenyls and polycyclic aromatic hydrocarbons in sediments from the lower Passaic River, New Jersey. *Arch. Environ. Contam. Toxicol.* 27:64-81.

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2.0 DATA COMPILATION AND EVALUATION

In this section, the available data on chemicals in sediments, surface water, and biota within the Site are compiled and evaluated. The primary objectives of the data evaluation were to (a) determine which data are most appropriate for use in the risk assessment, and (b) compile a preliminary list of chemicals of potential concern (CPC) for human and ecological receptors.

2.1 Sources of Sediment, Water Quality, and Biological Data

Sources of information on sediment and water quality stressors for the screening-level HERA include existing data on chemicals detected in sediments, water, and biota collected from the Site during both historic and recent sampling programs. Sediment data were evaluated from the existing datasets described on page 4-3 of the Investigation Work Plan (IWP), as well as additional datasets that were identified from a comprehensive data search for the Site (see page 2-7). Water quality data were derived from the EPA Storage and Retrieval of Water Quality Data (STORET) database and government reports. Biological data were derived from the scientific literature and government reports. Evaluation of biological community data is discussed in Section 4.2 (Characterization of the Ecological Community). Data on chemical concentrations in biota from the tidal Passaic River were included in the assessment of sediment and water quality stressors.

2.2 Data Quality Assessment

Available datasets (sediment, water, and biological) were evaluated utilizing relevant EPA guidance on data quality for risk assessment purposes (EPA, 1987, 1992), to determine whether the information contained therein should be included in the risk assessment. Datasets that were not evaluated and/or validated using EPA Region II Quality Assurance/Quality Control (QA/QC) protocols were flagged during the data compilation.

2.2.1 Data Grouping

Consistent with EPA guidance (EPA, 1989), the lateral and vertical distribution of chemicals in the Site media has been evaluated to determine the most appropriate definition of the exposure area(s) for the potentially exposed populations. This is necessary to ensure that the data are grouped properly. As described in the IWP, the Site has been defined laterally as one exposure area because aquatic organisms, such as fish and crabs, are mobile and, therefore, may be exposed to a range of chemical concentrations in surface sediments, water, and contaminated organisms (such as benthic invertebrates) throughout the Site and beyond. Likewise, humans may be exposed to fish and crabs that move throughout the Site and beyond. Therefore, for the purposes of this HERA, the Site is treated laterally as a single exposure area rather than discrete subunits.

With respect to the vertical definition of the Site, it is critical to note that fish and other aquatic organisms are only exposed to chemicals in surface sediments from the biologically active zone (BAZ) of the River. The BAZ, as defined in the AOC, comprises sediments from about 0 to 6 inches (i.e., about 0 to 15 cm) in depth. Sediments from the BAZ represent the most significant exposures of benthic organisms and higher food web organisms (i.e., fish and crabs) to chemicals in aquatic systems. Therefore, in summary, the Site sediments are treated as a single unit which extends laterally the length of the Site and extends vertically 6 inches into the surficial sediments. Chemical concentrations in these sediments are then used to estimate chemical concentrations in fish and other tissues using a food web model, as described in Section 4.0.

In addition to estimated tissue concentrations derived from the food web model, available data concerning actual measured tissue concentrations are considered in this HERA. Because fish and crabs are mobile, particularly the migratory species which represent the majority of the fish population at the Site (see Section 4.2.4.3), data on chemicals in biota were compiled for the entire tidal Passaic River. It is reasonable to expect that these data will give a better approximation of the range of chemical concentrations in biota that are moving throughout the River and beyond. This is particularly relevant for evaluating the risks from fish and crab ingestion to human consumers.

Summary statistics were calculated for chemical concentrations in surface sediments from the BAZ of the Site. The 95 percent upper confidence limit (UCL) of the arithmetic mean of the Site sediment data was used as representative chemical concentrations for the Site. Similarly, available biological (i.e., fish and crab tissue) data were summarized for the tidal Passaic River. The minimum, mean, and maximum chemical concentrations in various species of biota were calculated for use in the risk assessment. However, the biological dataset that was compiled was not sufficiently large to permit the calculation of confidence limits about the arithmetic mean of the data.

2.2.2 Sediment Data Evaluation Criteria

As described in the IWP, sediment data from different sampling programs were reviewed to evaluate their compatibility for use in the risk assessment through consideration of the following factors:

1. sample location;
2. depth of sediment sample collection;
3. date of sample collection;
4. data presentation (dry weight/wet weight);
5. analytical methods employed;
6. analytes detected;
7. quantitation/detection limits; and
8. analytical data quality (duplicates, blanks, spikes, etc.).

Sediment samples collected within the linear boundaries of the Site were considered useable for the screening-level HERA, including samples collected in small tidal tributaries of the Passaic River within the linear definition of the Site. The only requirement for inclusion into the data compilation was that the locations of the sample collections could be verified by either latitude-longitude coordinates, or an appropriate sampling location map.

The depth of each sediment sample from all datasets collected from the Site was evaluated. Samples or datasets that did not include sediments collected from the BAZ (i.e., 0 to 6 inches) at

the time of sample collection were determined to be unusable for the purposes of the screening-level HERA. Samples that included surface sediments, however, regardless of overall depth of the sample, were evaluated further.

Samples that were in the BAZ at the time of sample collection, but are presently buried below the BAZ, were determined to be unusable for the screening-level HERA. To assess whether historical surface sediment samples are still representative of the BAZ, sediment radiodating (^{210}Pb and ^{137}Cs) results from the Site were evaluated. From these data, sediment accumulation rates were estimated for the Site. Based on data from 26 sediment cores collected from the Site between 1991 and 1993, the mean sediment accumulation rate is estimated to be 1.7 in/yr. It is therefore estimated that surface sediments collected prior to about 1991 are likely to be buried below the BAZ. The results of several historical studies, including IT (1986), Suszkowski (1978), and Bopp et al. (1991) are consistent with these results. These studies reported that portions of the Site, particularly the left bank of the Harrison and Point-No-Point Reaches of the River, exhibit extremely high sediment accumulation rates. Radiodating of sediment cores collected in these Reaches of the River indicates that sediments have accumulated at an estimated rate of 2.6 in/yr. These results indicate that surface sediment samples collected prior to 1992 (in these Reaches of the River), are buried below the BAZ. However, because sediment accumulation rates are variable throughout the Site, and to ensure that all sediments which may be representative of the current BAZ are included in the risk assessment, samples collected between 1990 and 1994 were included in the screening-level HERA. This is consistent with the conservative approach to conducting the screening-level HERA. Datasets collected prior to 1990 were eliminated from further consideration since the sediments that they characterize are now buried below the BAZ.

Data compiled for use in the screening-level HERA were reported on a dry weight basis. Datasets are, therefore, comparable in this regard. However, the specific analytical methods employed were not always reported for all datasets. Less sensitive analytical methods would generally result in detection limits that are relatively high, as compared to more sensitive analytical methods. To be conservative, however, datasets analyzed using less sensitive analytical methods were not discarded, provided that they met the depth and data reporting criteria that were previously discussed.

Analytes that were not detected in any dataset were excluded from further evaluation. Analytes that were detected in one or more samples from any dataset from the Site were included for further evaluation. In cases where such an analyte was not detected in a particular sample, and either a sample detection limit or a method detection limit was reported, the analyte concentration was estimated for the sample as one-half the sample detection limit or as one-half the method detection limit. If both the sample detection limit and the method detection limit were reported, one-half the sample detection limit was used. If an analyte was not detected in a sample, and neither a sample detection limit nor method detection limit was reported, then that particular analyte in that sample was excluded from further evaluation. However, such an exclusion did not preclude the use of that sample for other chemicals for which either detected concentrations or either sample or method detection limits were reported.

QA/QC information typically includes the analytical results of field duplicate samples, field blanks, trip blanks, laboratory blanks, Matrix Spike/Matrix Spike Duplicates (MS/MSDs), and Matrix Spike and Laboratory Duplicates (MS/Ds). However, such QA/QC data were not available for most datasets and samples collected from the Site. In fact, none of the existing datasets have been evaluated using EPA Region II QA/QC protocols. For these reasons, all available data that met the criteria described above (i.e., the sample location is known and the sample was taken from what is currently the BAZ) were included in the dataset for the screening-level HERA. The only exceptions were those data that were rejected during data reviews or validations.

2.2.3 Biological Sample Data Evaluation

Data on chemical concentrations in biota from different sampling programs conducted in the tidal Passaic River were reviewed to evaluate their compatibility for use in the risk assessment. Biological datasets from the different sampling programs were evaluated for use in the risk assessment, as described in the IWP, through consideration of the following areas:

1. sample location;
2. date and season of sample collection;
3. analytical methods employed;
4. analytes detected;

5. quantitation/detection limits;
6. species analyzed;
7. tissues analyzed;
8. data reporting (dry weight/wet weight/lipid normalized); and
9. analytical quality (duplicates, blanks, spikes, etc.).

As previously discussed, available biological data from the tidal Passaic River were considered in the data evaluation. However, the available biological data were collected only between 1983 and 1988. Since these data were collected prior to 1990, they were determined to be unusable for evaluating current risks to ecological or human receptors. Given the limited quantity of biological data from the Site, and their limited use in the screening-level HERA, biological data were compiled and summarized regardless of the date or season of sample collection.

Numerous species of edible and non-edible fish and crustaceans have been collected in the tidal Passaic River, and analyzed for a limited number of chemicals in various tissues. These data were segregated by species into the tissues analyzed for the various species; these included whole body and edible (muscle) tissue in fish, and hepatopancreas, muscle, and whole body (e.g., hepatopancreas/muscle mixture) in crabs.

Biological data compiled for use in the screening-level HERA were reported on a wet weight basis. All datasets are, therefore, comparable in this regard. The majority of biological data were collected and analyzed by the New Jersey Department of Environmental Protection (NJDEP), thus, the analytical methods and data quality (including detection limits and QA/QC) are comparable. However, similar to the sediment data, none of these data were evaluated using EPA Region II QA/QC protocols.

2.2.4 Water Quality Data Evaluation

Very few data have been collected on water quality for the Site. Consequently, the available water quality data were not subjected to formal evaluation as was intended in the IWP. Rather, the data were simply compiled and sorted for qualitative evaluation in the ecological risk assessment.

2.3 Selection of Useable Datasets

The following sediment and biological datasets were evaluated for useability in the HERA:

	Dataset	Collection Date	Sediment Samples	Biological Samples
•	EPA (1984)	1983	x	
•	IT Corp. (1985)	1984	x	
•	IT Corp. (1986)	1984	x	
•	NJDEP (1985)	1983		x
•	EPA (1988)	1988	x	
•	USACE (1988)	1988	x	
•	USACE (1989)	1988	x	
•	NJDEP (1990)	1986		x
•	EPA (1992)	1986		x
•	Battelle (1992)	1992	x	
•	EPA (1993)	1993	x	
•	NJDEP (1993)	1988		x
•	OCC (1994)	1990/1991/1992/1993	x	
•	OCC (1995)	1994	x	

In total, ten different datasets on chemicals in sediments were available for evaluation. Of these ten datasets, six were determined to be unusable for the screening-level HERA because sediment samples were collected before 1990. The six datasets determined not to be useable are EPA (1984), IT (1985), USACE (1988), EPA (1988), USACE (1989), and IT (1986). The remaining four datasets were determined to be useable in the screening-level HERA, although individual samples or analytes may have been eliminated, as previously discussed. The final dataset on chemicals in surface sediments for the Site is presented in Appendix 1. Summary statistics for each chemical are provided in Table 2-1. None of the available datasets for the Site were evaluated using EPA Region II QA/QC protocols.

Table 2-1. Summary Statistics for Chemicals in Surface Sediments from the Passaic River Study Area

			Range (detected samples only)								Range of Detection Limits (non-detects only)	
Parameter	n	Freq. of Detection (%)	Minimum	Maximum	Mean	Median	Standard Deviation	Coefficient of Variation	95% Lower Confidence Level on the Mean	95% Upper Confidence Level on the Mean	Minimum	Maximum
PCDD/Fs (ng/kg)												
TCDD, 2,3,7,8-	51	100	2	1,600	340	270	300	0.88	260	420		
PeCDD, 1,2,3,7,8-	51	76	2.3	47	9.4	8.3	7.6	0.82	7.3	11	0.41	23
HxCDD, 1,2,3,4,7,8-	51	75	0.92	93	10	8.3	13	1.3	6.5	14	0.23	30
HxCDD, 1,2,3,6,7,8-	51	90	2.7	120	37	34	27	0.73	30	44	1	5
HxCDD, 1,2,3,7,8,9-	51	84	1.5	53	18	18	12	0.66	14	21	1	26
HpCDD, 1,2,3,4,6,7,8-	51	100	5.6	2,070	570	560	410	0.72	460	680		
OCDD	51	100	135	81,000	7,500	5,400	11,000	1.5	4,300	11,000		
Total TCDD	51	100	2	1,700	460	390	370	0.80	360	560		
Total PeCDD	51	88	4.4	1,190	100	66	190	1.9	49	150	1.8	9.22
Total HxCDD	51	94	7	1,100	320	280	260	0.81	250	390	5	5
Total HpCDD	51	100	20	5,890	1,300	1,200	1,000	0.83	970	1,500		
TCDF, 2,3,7,8-	51	98	1.8	280	39	27	48	1.2	26	52	0.66	0.66
PeCDF, 1,2,3,7,8-	51	90	1.5	580	28	17	80	2.8	6.5	50	0.25	5
PeCDF, 2,3,4,7,8-	51	90	4	1,400	80	52	190	2.4	27	130	0.4	5
HxCDF, 1,2,3,4,7,8-	51	98	8.6	20,000	610	170	2,800	4.6	0	1,400	0.97	0.97
HxCDF, 1,2,3,6,7,8-	51	98	2.6	2,900	110	53	400	3.6	2.4	220	0.33	0.33
HxCDF, 1,2,3,7,8,9-	51	84	0.54	300	14	6.9	41	2.9	2.8	25	0.42	18
HxCDF, 2,3,4,6,7,8-	51	88	2.8	780	48	32	110	2.3	18	77	0.56	5
HpCDF, 1,2,3,4,6,7,8-	51	100	2.6	64,000	2,100	870	8,900	4.2	0	4,500		
HpCDF, 1,2,3,4,7,8,9-	51	86	1.1	1,400	53	22	190	3.7	0	110	0.46	39
OCDF	51	98	50	130,000	3,800	1,200	18,000	4.8	0	8,700	4.1	4.1
Total TCDF	51	100	4.3	6,700	770	600	950	1.2	510	1,000		
Total PeCDF	51	100	4.8	11,000	850	650	1,500	1.8	440	1,300		
Total HxCDF	51	100	5.6	36,000	1,500	740	5,000	3.2	170	2,900		
Total HpCDF	51	100	2.6	76,000	2,800	1,300	10,000	3.8	0	5,700		

1/2 detection limit was used in calculations for samples that were non-detect.

Table 2-1 Summary Statistics for Chemicals in Surface Sediments from the Passaic River Study Area

			Range (detected samples only)								Range of Detection Limits (non-detects only)	
Parameter	n	Freq. of Detection (%)	Minimum	Maximum	Mean	Median	Standard Deviation	Coefficient of Variation	95% Lower Confidence Level on the Mean	95% Upper Confidence Level on the Mean	Minimum	Maximum
Acids (ug/kg)												
Methylphenol, 4-	46	2	140	140	560	460	390	0.71	440	670	420	3,800
Phenol	46	2	1,200	1,200	580	480	400	0.69	460	690	420	3,800

1/2 detection limits were used in calculations for samples that were non-detect.

Table 2-1. Summary Statistics for Chemicals in Surface Sediments from the Passaic River Study Area

			Range (detected samples only)								Range of Detection Limits (non-detects only)	
Parameter	n	Freq. of Detection (%)	Minimum	Maximum	Mean	Median	Standard Deviation	Coefficient of Variation	95% Lower Confidence Level on the Mean	95% Upper Confidence Level on the Mean	Minimum	Maximum
Bases (ug/kg)												
Bis(2-ethylhexyl)phthalate	46	98	960	43,000	15,000	14,000	10,000	0.67	12,000	18,000	840	3,800
Butyl benzyl phthalate	46	30	140	920	550	450	390	0.71	440	670	430	3,800
Di-n-butyl phthalate	46	7	230	820	590	480	400	0.69	470	710	420	3,800
Di-n-octyl phthalate	46	48	110	5,000	680	480	770	1.1	460	900	420	3,800
Dichlorobenzene, 1,4-	46	17	130	1,800	590	480	430	0.72	470	720	430	3,800
Dimethylphthalate	46	2	1,100	1,100	570	470	400	0.69	460	690	420	3,800
Trichlorobenzene, 1,2,4-	46	2	2,500	2,500	610	480	480	0.79	470	750	420	3,800

1/2 detection limits were used in all calculations for samples that were non-detect.

Table 2-1. Summary Statistics for Chemicals in Surface Sediments from the Passaic River Study Area

			Range (detected samples only)								Range of Detection Limits (non-detects only)	
Parameter	n	Freq. of Detection (%)	Minimum	Maximum	Mean	Median	Standard Deviation	Coefficient of Variation	95% Lower Confidence Level on the Mean	95% Upper Confidence Level on the Mean	Minimum	Maximum
Metals (mg/kg)												
Aluminum	47	100	4,550	24,100	13,100	14,500	5,240	0.4	11,600	14,600		
Antimony	47	11	15.6	39.6	7.9	6.3	8.1	1.0	5.6	10	0.2	27.9
Arsenic	45	96	3.3	62.3	13	12	8.9	0.70	10	15	1.6	8.1
Barium	47	100	33.7	1,280	179	154	173	0.968	130	229		
Beryllium	47	96	0.3	3.1	1.0	0.82	0.67	0.65	0.85	1.2	0.28	0.29
Cadmium	48	98	0.76	14	6.3	6.3	3.0	0.47	5.5	7.2	0.54	0.54
Calcium	47	100	1,130	14,600	6,520	6,250	2,540	0.390	5,790	7,250		
Chromium	47	100	25.8	402	158	167	70.7	0.447	138	179		
Cobalt	47	100	5.6	41.1	14	12	6.3	0.46	12	15		
Copper	46	100	26.4	437	237	239	78.4	0.331	214	260		
Cyanide	37	24	0.29	269	9.3	0.70	44	4.7	0	24	0.6	2.4
Iron	47	100	15,100	43,900	28,400	28,700	6,450	0.227	26,600	30,200		
Lead	46	100	31.3	840	359	346	123	0.342	324	395		
Magnesium	47	100	2,820	11,100	6,210	6,410	2,060	0.332	5,620	6,800		
Manganese	45	100	134	875	383	403	162	0.423	336	430		
Mercury	48	98	0.57	8.1	3.4	3.3	1.8	0.52	2.9	3.9	0.17	0.17
Nickel	48	100	16.8	178	57.3	52.7	28.4	0.496	49.3	65.4		
Potassium	47	100	493	4,710	2,080	2,060	973	0.468	1,800	2,360		
Selenium	47	34	0.78	3.3	1.2	0.80	1.2	0.98	0.88	1.6	0.67	11.4
Silver	48	81	1.2	39.5	5.3	4.2	6.2	1.2	3.5	7.1	0.81	3.3
Sodium	47	100	461	14,800	5,580	4,060	3,750	0.672	4,500	6,650		
Thallium	47	13	0.25	1.9	0.52	0.38	0.38	0.73	0.41	0.63	0.35	2.4
Titanium	14	100	212	605	420	453	127	0.303	346	493		
Vanadium	47	100	18.7	80.6	39.6	41.9	11.9	0.300	36.2	43.0		
Zinc	46	100	76.6	1,060	575	569	182	0.317	522	628		

1/2 detection limits were used in all calculations for samples that were non-detect.

Table 2-1. Summary Statistics for Chemicals in Surface Sediments from the Passaic River Study Area

Parameter	n	Freq. of Detection (%)	Range (detected samples only) Minimum	Range (detected samples only) Maximum	Mean	Median	Standard Deviation	Coefficient of Variation	95% Lower Confidence Level on the Mean	95% Upper Confidence Level on the Mean	Range of Detection Limits (non-detects only) Minimum	Range of Detection Limits (non-detects only) Maximum
PCBs (ug/kg)												
TetraCB, 3,3',4,4'- (IUPAC #77)	46	100	0.018	86	9.0	6.7	13	1.4	5.3	13		
PentaCB, 2',3,4,4',5- (IUPAC #123)	20	100	0.67	7.1	4.1	4.0	2.1	0.50	3.2	5.1		
PentaCB, 2,3',4,4',5- (IUPAC #118)	46	100	0.13	320	43	35	47	1.1	30	57		
PentaCB, 2,3,3',4,4'- (IUPAC #105)	46	100	0.052	190	19	16	27	1.4	11	27		
PentaCB, 2,3,4,4',5- (IUPAC #114)	20	100	0.17	2.4	1.3	1.3	0.63	0.48	1.0	1.6		
PentaCB, 3,3',4,4',5- (IUPAC #126)	46	87	0.035	2	0.29	0.21	0.32	1.1	0.20	0.38	0.00071	2
HexaCB, 2,3',4,4',5,5'- (IUPAC #167)	20	100	1.1	14	7.6	8.2	3.3	0.44	6.0	9.1		
HexaCB, 2,3,3',4,4',5'- (IUPAC #157)	20	100	0.18	3.5	1.5	1.5	0.77	0.52	1.1	1.8		
HexaCB, 2,3,3',4,4',5- (IUPAC #156)	20	100	0.65	9.6	4.7	5.0	2.2	0.47	3.7	5.8		
HexaCB, 3,3',4,4',5,5'- (IUPAC #169)	46	30	0.0051	0.078	0.018	0.012	0.018	1.0	0.013	0.024	0.0024	0.15
HeptaCB, 2,3,3',4,4',5,5'- (IUPAC #189)	20	100	0.14	4.3	1.8	1.8	1.2	0.65	1.2	2.3		
Aroclor 1248	47	60	53.5	6,020	548	305	939	1.71	279	816	20	819
Aroclor 1254	47	13	485	918	139	43.0	216	1.55	77.3	201	20	919

1/2 detection limit was used in calculations for samples that were non-detect.

Table 2-1. Summary Statistics for Chemicals in Surface Sediments from the Passaic River Study Area

			Range (detected samples only)								Range of Detection Limits (non-detects only)	
Parameter	n	Freq. of Detection (%)	Minimum	Maximum	Mean	Median	Standard Deviation	Coefficient of Variation	95% Lower Confidence Level on the Mean	95% Upper Confidence Level on the Mean	Minimum	Maximum
Pesticides (ug/kg)												
Aldrin	47	28	4.81	59.8	7.7	2.3	11	1.4	4.6	11	1	47.3
alpha-Chlordane	46	70	3.5	66	17	16	13	0.79	13	21	1.99	47.3
Beta-BHC	47	9	3.14	56.2	4.46	2.12	9.00	2.02	1.88	7.03	1.99	47.3
Chlordane	1	100	18.0	18.0	18.0	18.0	NA	NA	NA	NA		
DDD, 4,4'-	47	89	5.59	591	109	55.0	144	1.32	68.0	150	3.86	91.9
DDE, 4,4'-	47	83	11.5	106	42.7	41.0	25.9	0.607	35.3	50.1	3.86	91.9
DDT, 4,4'-	47	66	6.19	293	37	18	58	1.6	20	53	3	91.9
Delta-BHC	47	15	4.67	23.8	4.42	2.16	5.9	1.34	2.73	6.10	1.99	47.3
Dieldrin	47	34	7.93	270	17	5.3	39	2.4	5.3	28	3	91.9
Endosulfan I	47	2	12	12	3.64	2.11	5.24	1.44	2.14	5.13	1.99	47.3
Endosulfan II	47	45	7.89	123	21.2	8.41	24.9	1.17	14.1	28.3	3.86	91.9
Endosulfan sulfate	47	6	8.51	9.46	6.93	4.23	9.85	1.42	4.11	9.74	3.86	91.9
Endrin	47	30	19	134	19.8	5.00	26.6	1.34	12.2	27.4	3.86	91.9
Endrin aldehyde	47	19	5.9	38.5	8.3	4.5	11	1.3	5.2	11	2	91.9
Endrin ketone	46	30	7.4	82.7	17.8	4.73	21.5	1.20	11.6	24.1	3.86	91.9
gamma-Chlordane	46	78	3.39	117	18.8	15.6	18.8	1.00	13.3	24.2	1.99	47.3
Heptachlor epoxide (exo)	12	17	4.25	12.9	2.92	2.05	3.25	1.11	0.860	4.99	1.99	4.83
Methoxychlor	47	6	32.7	445	35	21	69	2.0	16	55	3	422

1/2 detection limit was used in calculations for samples that were non-detect.
NA: Not applicable

Table 2-1. Summary Statistics for Chemicals in Surface Sediments from the Passaic River Study Area

			Range (detected samples only)								Range of Detection Limits (non-detects only)	
Parameter	n	Freq. of Detection (%)	Minimum	Maximum	Mean	Median	Standard Deviation	Coefficient of Variation	95% Lower Confidence Level on the Mean	95% Upper Confidence Level on the Mean	Minimum	Maximum
Volatile Organic Compounds (ug/kg)												
Acetone	31	94	8	14,000	1,300	70	3,100	2.4	190	2,400	14	15
Benzene	31	6	7	17	12	13	3.9	0.33	10	13	13	38
Butanone, 2-	31	29	9	64	16	14	11	0.72	12	20	13	38
Chlorobenzene	31	32	7	1,400	110	14	330	3.0	0	230	13	35
Chloromethane	31	13	3	48	12	12	7.7	0.62	9.7	15	13	35
Dichloroethene, 1,2- (total)	31	6	13	20	12	13	3.7	0.32	10	13	13	35
Ethyl Benzene	31	10	4	76	14	13	12	0.91	9.2	18	13	38
Methylene Chloride	31	19	3	37	12	11	6.4	0.53	9.9	14	13	38
Toluene	31	16	4	100	18	13	21	1.2	10	25	13	35
Xylene (total)	31	13	14	440	27	13	77	2.8	0.16	55	13	38

1/2 detection limits were used in all calculations for samples that were non-detect.

Table 2-1. Summary Statistics for Chemicals in Surface Sediments from the Passaic River Study Area

			Range (detected samples only)								Range of Detection Limits (non-detects only)	
Parameter	n	Freq. of Detection (%)	Minimum	Maximum	Mean	Median	Standard Deviation	Coefficient of Variation	95% Lower Confidence Level on the Mean	95% Upper Confidence Level on the Mean	Minimum	Maximum
PAHs (ug/kg)												
Acenaphthene	46	20	230	3,800	710	480	660	0.94	510	900	420	3,800
Acenaphthylene	46	52	140	1,000	540	420	410	0.75	420	660	420	3,800
Anthracene	45	84	87	5,100	820	480	950	1.2	540	1,100	420	3,800
Benzo(a)anthracene	46	91	300	5,800	1,600	1,250	1,200	0.77	1,200	1,900	840	3,800
Benzo(a)pyrene	46	93	300	4,300	1,800	1,650	890	0.50	1,500	2,000	840	3,500
Benzo(b)fluoranthene	46	96	310	4,300	1,800	1,650	940	0.53	1,500	2,000	840	3,800
Benzo(ghi)perylene	46	93	170	2,500	1,100	1,100	590	0.52	970	1,300	840	3,800
Benzo(k)fluoranthene	46	96	200	6,300	1,700	1,600	1,200	0.70	1,400	2,000	840	3,800
Carbazole	45	33	120	1,400	600	480	430	0.72	470	720	420	3,800
Chrysene	46	98	340	5,900	1,800	1,500	1,200	0.64	1,500	2,200	840	3,800
Dibenzo(a,h)anthracene	46	52	140	1,500	640	490	410	0.64	520	760	420	3,800
Dibenzofuran	46	13	250	3,000	620	470	530	0.85	470	780	420	3,800
Fluoranthene	46	100	660	11,000	3,500	3,000	2,400	0.69	2,800	4,200	840	3,800
Fluorene	46	20	180	4,300	680	480	680	1.0	480	880	420	3,800
Indeno(1,2,3-c,d)pyrene	46	98	200	2,500	1,200	1,100	610	0.51	1,000	1,400	840	3,800
Methylnaphthalene, 2-	46	9	160	4,300	660	480	680	1.0	460	850	420	3,800
Naphthalene	46	11	550	6,500	790	490	1,000	1.3	490	1,100	420	3,800
Phenanthrene	46	98	210	14,000	1,900	1,100	2,500	1.3	1,100	2,600	840	3,800
Pyrene	46	100	630	11,000	3,200	2,600	2,300	0.71	2,600	3,900	840	3,800
High Molecular Weight (a)	46	100	2,500	50,000	18,000	16,000	11,000	0.57	15,000	21,000	420	3,800
Low Molecular Weight (b)	46	98	210	42,000	7,200	5,200	6,900	0.95	5,300	9,200	420	3,800

1/2 detection limits were used in all calculations for samples that were non-detect.
(a) Sum of all PAHs with four or more rings.
(b) Sum of all PAHs with two or three rings.

Table 2-1. Summary Statistics for Chemicals in Surface Sediments from the Passaic River Study Area

Parameter	n	Freq. of Detection (%)	Range (detected samples only) Minimum	Range (detected samples only) Maximum	Mean	Median	Standard Deviation	Coefficient of Variation	95% Lower Confidence Level on the Mean	95% Upper Confidence Level on the Mean	Range of Detection Limits (non-detects only) Minimum	Range of Detection Limits (non-detects only) Maximum
TEPH (mg/kg)	46	96	30	2,740	875	504	858	0.981	627	1,120	28.4	74.4
Total Organic Carbon (mg/kg)	46	100	370.1	233,000	58,700	38,900	55,700	0.949	42,600	74,800	10	10
Dibutyltin (ug/kg)	10	10	742	742	193	168	199	1.03	50.5	335	94.9	363
Monobutyltin (ug/kg)	10	20	276	835	328	328	201	0.611	185	471	185	727

1/2 detection limits were used in all calculations for samples that were non-detect.

A total of four different datasets on chemicals in biota were available for evaluation. Generally, all four datasets were determined to be unusable for assessing risk, as previously discussed. Nonetheless, the data are considered in this HERA for qualitative comparison purposes. The final dataset on chemicals in biota (as compiled from the four existing datasets) from the tidal Passaic River is presented in Table 2-2.

The limited water quality data that are available for the Site are presented in Table 2-3. As previously discussed, these data will only be used for qualitative purposes in the ecological risk assessment.

2.4 Preliminary List of Chemicals of Potential Concern

Data determined to be of sufficient quality for use in risk assessment were compiled and summarized, as previously described. Consistent with EPA guidance (EPA, 1992), and the criteria identified in the IWP, the preliminary CPC comprise all analytes detected in sediments, including inorganic chemicals and organic compounds that were detected in surface sediments from any of the existing samples that were included in the final dataset for the screening-level HERA.

The list of preliminary CPC is provided in Table 2-4. These data are further evaluated in Sections 3.2 and 4.3 to determine the final lists of human health and ecological CPC, respectively.

Table 2-2a. Available Chemical Data for Biota Samples Collected from the Tidal Passaic River, New Jersey

Sampling Site	Species		Aroclor 1248	Aroclor 1254/60	Total PCBs	alpha chlordane	gamma chlordane	Total chlordane	DDT	DDD	DDE	Total DDTs	2,3,7,8-TCDD	2,3,7,8-TCDF		Reference
		Lipids	ppm	ppm	ppm	ppb	ppb	ppb	ppb	ppb	ppb	ppb	ppt	ppt	Sampling	
		%	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	Year	
4th St. Bridge, Harrison	American eel												20.00		1982	NJDEP, 1985
4th St. Bridge, Harrison	American eel												31.00		1982	NJDEP, 1985
4th St. Bridge, Harrison	American eel												61.00		1983	NJDEP, 1985
4th St. Bridge, Harrison	American eel												22.00		1983	NJDEP, 1985
4th St. Bridge, Harrison	American eel												31.00		1983	NJDEP, 1985
Avondale Swing Bridge, Lyndhurst	American eel												56.50		1983	NJDEP, 1985
Carlton Hills, Rutherford	American eel												80.00		1983	NJDEP, 1985
Confluence w/Newark Bay	American eel	2.55	0.28	0.37	0.65	26.21	10.08	36.29	18.38	115.74	116.28	250.40			1986	NJDEP, 1990
Monroe St. Bridge	American eel	7.50	1.25	1.21	2.46	165.44	29.26	194.70	29.17	104.17	120.90	254.24			1988	NJDEP, 1993
Unknown	American eel				7.18										1981	NJDEP, 1983
	N	2	2	2	3	2	2	2	2	2	2	2	7			
	Minimum	**2.55**	**0.28**	**0.37**	**0.65**	**26.21**	**10.08**	**36.29**	**18.38**	**104.17**	**116.28**	**250.40**	**20.00**			
	Maximum	**7.50**	**1.25**	**1.21**	**7.18**	**165.44**	**29.26**	**194.70**	**29.17**	**115.74**	**120.90**	**254.24**	**80.00**			
	Mean	**5.03**	**0.77**	**0.79**	**3.43**	**95.83**	**19.67**	**115.50**	**23.78**	**109.96**	**118.59**	**252.32**	**43.07**			
80 Lister Ave.	Blue crab (H)(a)												485.00		1983	NJDEP, 1985
80 Lister Ave.	Blue crab (H)												450.00		1983	NJDEP, 1985
Confluence w/Newark Bay	Blue crab (H)	7.54	1.79	1.90	3.69	20.38	27.64	48.02	30.84	154.07	184.48	369.39			1988	NJDEP, 1993
Confluence w/Newark Bay	Blue crab (H)	6.97	2.58	3.71	6.29	84.56	27.57	112.13	27.57	201.61	285.00	514.18			1987	NJDEP, 1990
	N	2	2	2	2	2	2	2	2	2	2	2	2			
	Minimum	**6.97**	**1.79**	**1.90**	**3.69**	**20.38**	**27.57**	**48.02**	**27.57**	**154.07**	**184.48**	**369.39**	**450.00**			
	Maximum	**7.54**	**2.58**	**3.71**	**6.29**	**84.56**	**27.64**	**112.13**	**30.84**	**201.61**	**285.00**	**514.18**	**485.00**			
	Mean	**7.26**	**2.19**	**2.81**	**4.99**	**52.47**	**27.61**	**80.08**	**29.21**	**177.84**	**234.74**	**441.79**	**467.50**			
80 Lister Ave.	Blue crab (H/M)												480.00		1983	NJDEP, 1985
Confluence w/Newark Bay	Blue crab (H/M)	1.04	0.76	0.61	1.37	15.03	8.82	23.85	5.00	43.75	61.42	110.17			1988	NJDEP, 1993
Confluence w/Newark Bay	Blue crab (H/M)	1.85	0.58	0.60	1.18	15.96	3.27	19.23	5.00	24.80	47.24	77.04			1986	NJDEP, 1990
Confluence w/Newark Bay	Blue crab (H/M)	10.36	0.93	0.86	1.79	23.24	4.59	27.83	10.73	75.73	107.55	194.01			1986	NJDEP, 1990
Confluence w/Newark Bay	Blue crab (H/M)	2.32	0.88	0.99	1.87	15.20	8.15	23.35	5.00	52.88	115.91	173.79			1987	NJDEP, 1990
Confluence w/Newark Bay	Blue crab (H/M)	2.74	0.98	1.07	2.05	37.09	8.43	45.52	10.70	76.99	129.31	217.00			1988	NJDEP, 1993
Confluence w/Newark Bay	Blue crab (H/M)	2.25	1.11	2.23	3.34	71.43	11.03	82.46	5.00	139.16	227.50	371.66			1986	NJDEP, 1990
	N	6	6	6	6	6	6	6	6	6	6	6	1			
	Minimum	**1.04**	**0.58**	**0.60**	**1.18**	**15.03**	**3.27**	**19.23**	**5.00**	**24.80**	**47.24**	**77.04**	**480.00**			
	Maximum	**10.36**	**1.11**	**2.23**	**3.34**	**71.43**	**11.03**	**82.46**	**10.73**	**139.16**	**227.50**	**371.66**	**480.00**			
	Mean	**3.43**	**0.87**	**1.06**	**1.93**	**29.66**	**7.38**	**37.04**	**6.91**	**68.89**	**114.82**	**190.61**	**480.00**			
80 Lister Ave.	Blue crab (M)												27.00		1983	NJDEP, 1985
80 Lister Ave.	Blue crab (M)												16.00		1983	NJDEP, 1985
Confluence w/Newark Bay	Blue crab (M)	0.76	0.19	0.25	0.44	4.76	2.52	7.28	5.00	16.25	22.58	43.83			1988	NJDEP, 1993
Confluence w/Newark Bay	Blue crab (M)	0.83	0.21	0.18	0.39	6.25	4.04	10.29	5.00	19.66	32.75	57.41			1987	NJDEP, 1990
	N	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00			
	Minimum	**0.76**	**0.19**	**0.18**	**0.39**	**4.76**	**2.52**	**7.28**	**5.00**	**16.25**	**22.58**	**43.83**	**16.00**			
	Maximum	**0.83**	**0.21**	**0.25**	**0.44**	**6.25**	**4.04**	**10.29**	**5.00**	**19.66**	**32.75**	**57.41**	**27.00**			
	Mean	**0.80**	**0.20**	**0.22**	**0.42**	**5.51**	**3.28**	**8.79**	**5.00**	**17.96**	**27.67**	**50.62**	**21.50**			

Table 2-2a. Available Chemical Data for Biota Samples Collected from the Tidal Passaic River, New Jersey

Sampling Site	Species		Aroclor 1248	Aroclor 1254/60	Total PCBs	alpha chlordane	gamma chlordane	Total chlordane	DDT	DDD	DDE	Total DDTs	2,3,7,8-TCDD	2,3,7,8-TCDF		Reference
		Lipids	ppm	ppm	ppm	ppb	ppb	ppb	ppb	ppb	ppb	ppb	ppt	ppt	Sampling	
		%	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	wet weight	Year	
4th St. Bridge, Harrison	Brown bullhead												73.00		1983	NJDEP, 1985
Carlton Hills, Rutherford	Brown bullhead												110.00		1983	NJDEP, 1985
	N												**2.00**			
	Minimum												**73.00**			
	Maximum												**110.00**			
	Mean												**91.50**			
Lyndhurst	Carp												100.00		1983	NJDEP, 1985
Carlton Hills, Rutherford	Carp												210.00		1983	NJDEP, 1985
Confluence w/Third River	Carp												155.00		1983	NJDEP, 1985
Monroe St. Bridge	Carp	4.53	1.40	1.59	2.99	117.65	83.78	201.43	5.00	153.98	125.00	283.98			1988	NJDEP, 1993
Monroe St. Bridge	Carp	5.30	3.13	2.70	5.83	364.46	219.70	584.16	13.30	390.63	401.16	805.09			1986	NJDEP, 1990
Monroe St., Wallington	Carp												108.00		1983	NJDEP, 1985
	N	**2.00**	**2.00**	**2.00**	**2.00**	**2.00**	**2.00**	**2.00**	**2.00**	**2.00**	**2.00**	**2.00**	**4.00**			
	Minimum	**4.53**	**1.40**	**1.59**	**2.99**	**117.65**	**83.78**	**201.43**	**5.00**	**153.98**	**125.00**	**283.98**	**100.00**			
	Maximum	**5.30**	**3.13**	**2.70**	**5.83**	**364.46**	**219.70**	**584.16**	**13.30**	**390.63**	**401.16**	**805.09**	**210.00**			
	Mean	**4.92**	**2.27**	**2.15**	**4.41**	**241.06**	**151.74**	**392.80**	**9.15**	**272.31**	**263.08**	**544.54**	**143.25**			
4th St. Bridge, Harrison	Mummichog												114.00		1983	NJDEP, 1985
Lyndhurst	White perch												38.00		1983	NJDEP, 1985
Below Dundee Dam	White perch												5.00		1984	NJDEP, 1985
Unknown	White perch				3.72											
	N												**2.00**			
	Minimum												**5.00**			
	Maximum												**38.00**			
	Mean												**21.50**			
Confluence w/Newark Bay	Striped bass	1.86	0.50	0.66	1.16	16.65	6.12	22.77	5.00	56.73	60.33	122.06			1986	NJDEP, 1990
Confluence w/Newark Bay	Striped bass	3.23	1.70	2.36	4.06	34.65	12.15	46.80	5.00	15.12	104.16	124.28			1987	NJDEP, 1990
Confluence w/Newark Bay	Striped bass												23.00	8.00	1983	NJDEP, 1985
Confluence w/Newark Bay	Striped bass												56.00	33.00	1983	NJDEP, 1985
Confluence w/Newark Bay	Striped bass												32.00	29.00	1983	NJDEP, 1985
Confluence w/Newark Bay	Striped bass												47.00	42.00	1983	NJDEP, 1985
Confluence w/Newark Bay	Striped bass												58.00		1982	NJDEP, 1985
Confluence w/Newark Bay	Striped bass												31.00		1982	NJDEP, 1985
Confluence w/Newark Bay	Striped bass	1.03	0.78	0.61	1.39	28.09	15.98	44.07	5.00	55.02	62.50	122.52			1988	NJDEP, 1993
Confluence w/Newark Bay	Striped bass	3.00	1.47	1.01	2.48	31.69	25.24	56.93	26.12	54.10	78.13	158.35			1988	NJDEP, 1993
Unknown	Striped bass				6.04										1981	NJDEP, 1983
	N	**4.00**	**4.00**	**4.00**	**5.00**	**4.00**	**4.00**	**4.00**	**4.00**	**4.00**	**4.00**	**4.00**	**6.00**	**4.00**		
	Minimum	**1.03**	**0.50**	**0.61**	**1.16**	**16.65**	**6.12**	**22.77**	**5.00**	**15.12**	**60.33**	**122.06**	**23.00**	**8.00**		
	Maximum	**3.23**	**1.70**	**2.36**	**6.04**	**34.65**	**25.24**	**56.93**	**26.12**	**56.73**	**104.16**	**158.35**	**58.00**	**42.00**		
	Mean	**2.28**	**1.11**	**1.16**	**3.03**	**27.77**	**14.87**	**42.64**	**10.28**	**45.24**	**76.28**	**131.80**	**41.17**	**28.00**		

Shaded values are one half the reported detection limits for non-detect samples
(a) Blue Crab data are reported for hepatopancreas only (H), hepatopancreas/muscle mixture (H/M), and muscle only (M)

Table 2-2b. Chemical Concentrations in a Single Bluefish Sample Collected from the Passaic River Study Area During the National Bioaccumulation Study, 1986

PCDD/Fs (ppb)

Sampling Site	2,3,7,8-TCDD	1,2,3,7,8-PeDD	1,2,3,4,7,8-HxDD	1,2,3,6,7,8-HxDD	1,2,3,7,8,9-HxDD	1,2,3,4,6,7,8-HpDD	2,3,7,8-TCDF	1,2,3,7,8-PeDF	2,3,4,7,8-PeDF	1,2,3,4,7,8-HxDF	1,2,3,6,7,8-HxDF	1,2,3,7,8,9-HxDF	2,3,4,6,7,8-HxDF	1,2,3,4,6,7,8-HpDF	1,2,3,4,7,8,9-HpDF
Passaic River - Harrison Reach	ND	ND	ND	ND	ND	ND	0.0018	ND	0.00098	ND	ND	ND	ND	ND	ND
Detection limit	0.001	0.001	0.0025	0.0018	0.0014	0.0013		0.00087		0.0028	0.0028	0.0028	0.0019	0.0014	0.0026

Pesticides (ppb)

Sampling Site	alpha BHC	gamma BHC (lindane)	cis-Chlordane	trans-Chlordane	Oxy chlordane	trans-Nonachlor	Heptachlor	Heptachlor epoxide	DDE	Dieldrin	Endrin	Methoxychlor	Mirex
Passaic River - Harrison Reach	ND	ND	8.36	3.61	ND	11.6	ND	ND	60.2	4.47	ND	ND	ND
Detection limit	2.5	2.5			2.5		2.5	2.5			2.5	2.5	2.5

PCBs (ppb)

Sampling Site	Total PCBs	Total MonoCBs	Total DiCBs	Total TriCBs	Total TetraCBs	Total PentaCBs	Total HexaCBs	Total HectaCBs	Total OctaCBs	Total NonaCBs	Total DecaCBs
Passaic River - Harrison Reach	697.8	ND	ND	24.4	246	260	151	16.4	ND	ND	ND
Detection limit		1.25	1.25						3.75	6.25	6.25

Other Chemicals

Sampling Site	1,2,3-trichloro benzene (ppb)	1,2,4-trichloro benzene (ppb)	Mercury (ppm)
Passaic River - Harrison Reach	0.64	0.72	0.19
Detection limit			

Table 2-3. Available Water Quality Data from the Passaic River Study Area

SOURCE	IT, 1986	NJDEP, 1990	NOAA, 1985	STORET	STORET	US DOI, 1969	US DOI, 1969	US DOI, 1969	US DOI, 1969	US DOI, 1969	US DOI, 1969	ChemRisk, 1995
SAMPLING LOCATIONS	Lower Passaic River	Lower Passaic River	Lower Passaic River	2nd River at Belleville, NJ	3rd River, Nutley, NJ	Mile 0, Passaic River	Mile 1, Passaic River	Mile 2, Passaic River	Mile 3, Passaic River	Mile 4, Passaic River	Mile 5, Passaic River	Passaic River Study Area
DATE SAMPLED	Nov 1985		pre-1974	1962-1963	1963-1965	1969	1969	1969	1969	1969	1969	1994
WATER QUALITY PARAMETERS												
Biological												
Fecal coliform, geometric mean number/100mL		40-2,710				2,100	68,000	40,000	52,000	38,000	5,100	
Total coliform, number in more than 20% of samples/100 mL						17,000	500,000	400,000	340,000	300,000	42,000	
Chemical												
Gases												
Dissolved oxygen, mg/L	4.59- 8.10	>4.0	<3.0			1.4	1.0	2.4	0.0	0.8	1.3	2.2 - 5.8
Miscellaneous												
Salinity, ppth												6.0 - 23.0
pH, unitless	6.84- 7.67			7.1-8.2	6.9-8	7.3	7.1	7.2	7.2	7.3	7.3	
Phosphorus, total, ug/L		360-440										

Table 2-4. Preliminary Chemicals of Potential Concern in Sediments from the Passaic River Study Area

PCDD/Fs	Acids/Bases	Metals	PCBs	Pesticides	Volatile Organic Compounds	PAHs	Other
TCDD, 2,3,7,8-	Bis(2-ethylhexyl)phthalate	Aluminum	TetraCB, 3,3',4,4'- (IUPAC #77)	Aldrin	Acetone	Acenaphthene	TEPH
PeCDD, 1,2,3,7,8-	Butyl benzyl phthalate	Antimony	PentaCB, 2',3,4,4',5-(IUPAC #123)	alpha-Chlordane	Benzene	Acenaphthylene	Dibutyltin (ug/kg)
HxCDD, 1,2,3,4,7,8-	Di-n-butyl phthalate	Arsenic	PentaCB, 2,3',4,4',5- (IUPAC #118)	beta-BHC	Butanone, 2-	Anthracene	Monobutyltin (ug/kg)
HxCDD, 1,2,3,6,7,8-	Di-n-octyl phthalate	Barium	PentaCB, 2,3,3',4,4'- (IUPAC #105)	Chlordane	Chlorobenzene	Benzo(a)anthracene	
HxCDD, 1,2,3,7,8,9-	Dichlorobenzene, 1,4-	Beryllium	PentaCB, 2,3,4,4',5-(IUPAC #114)	DDD, 4,4'-	Chloromethane	Benzo(a)pyrene	
HpCDD, 1,2,3,4,6,7,8-	Dimethylphthalate	Cadmium	PentaCB, 3,3',4,4',5- (IUPAC #126)	DDE, 4,4'-	Dichloroethene, 1,2- (total)	Benzo(b)fluoranthene	
OCDD	Trichlorobenzene, 1,2,4-	Calcium	HexaCB, 2,3',4,4',5,5'-(IUPAC #167)	DDT, 4,4'-	Ethyl benzene	Benzo(ghi)perylene	
TCDF, 2,3,7,8-	Methylphenol, 4-	Chromium	HexaCB, 2,3,3',4,4',5'-(IUPAC #156)	delta-BHC	Methylene chloride	Benzo(k)fluoranthene	
PeCDF, 1,2,3,7,8-	Phenol	Cobalt	HexaCB, 2,3,3',4,4',5-(IUPAC #157)	Dieldrin	Toluene	Carbazole	
PeCDF, 2,3,4,7,8-		Copper	HexaCB, 3,3',4,4',5,5'- (IUPAC #169)	Endosulfan I	Xylene (total)	Chrysene	
HxCDF, 1,2,3,4,7,8-		Cyanide	HeptaCB, 2,3,3',4,4',5,5'-(IUPAC #189)	Endosulfan II		Dibenzo(a,h)anthracene	
HxCDF, 1,2,3,6,7,8-		Iron	Aroclor -1248	Endosulfan sulfate		Dibenzofuran	
HxCDF, 1,2,3,7,8,9-		Lead	Aroclor -1254	Endrin		Fluoranthene	
HxCDF, 2,3,4,6,7,8-		Magnesium		Endrin aldehyde		Fluorene	
HpCDF, 1,2,3,4,6,7,8-		Manganese		Endrin ketone		Indeno(1,2,3-c,d)pyrene	
HpCDF, 1,2,3,4,7,8,9-		Mercury		gamma-Chlordane		Methylnaphthalene, 2-	
OCDF		Nickel		Heptachlor epoxide (exo)		Naphthalene	
		Potassium		Methoxychlor		Phenanthrene	
		Selenium				Pyrene	
		Silver					
		Sodium				High Molecular Weight PAHs	
		Thallium				Low Molecular Weight PAHs	
		Titanium					
		Vanadium					
		Zinc					

2.5 References for Section 2.0

Battelle. 1992. *Sediment Toxicity and Concentrations of Trace Metals in Sediment and Porewater in New York/New Jersey Harbor, Data Report.* Prepared by Battelle Ocean Sciences, Duxbury, MA for New York City Department of Environmental Protection. June 8.

Bopp, R.F. and H.J. Simpson. 1991. *Sediment Sampling and Radionuclide and Chlorinated Hydrocarbon Analysis in Newark Bay and the Hackensack and Passaic Rivers: Final Report.* State of New Jersey Department of Environmental Protection, Division of Science and Research. Columbia University, Lamont-Doherty Geological Observatory. March 31.

EPA. 1984. USEPA Region II FIT Team Data - January. In: *Analytical Data Summary Tables Related to Passaic River Study.* Presented to U.S. Environmental Protection Agency, Region II, NY and New Jersey Department of Environmental Protection, April 22, 1993.

EPA. 1987. *Data Quality Objectives for Remedial Response Activities: Example Scenario: (RI/FS Activities at a Site with Contaminated Soils and Ground Water).* Prepared by CDM Federal Programs Corporation, Annandale, VA for the U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Office of Waste Programs Enforcement, Washington, D.C. PB88-131388. March.

EPA. 1988. Unpublished Data.

EPA. 1989. *Risk Assessment Guidance for Superfund; Volume I: Human Health Evaluation Manual (Part A) - Interim Final.* U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-89-002. July.

EPA. 1992. *National Study of Chemical Residues in Fish. Volume II.* U.S. Environmental Protection Agency, Office of Science and Technology, Washington, DC. EPA 823-R-92-008b. September.

EPA. 1993. Memorandum from L. Richman, Remedial Project Manager to R. McNutt. Re: EPA/NOAA Recent Data with Laboratory Data Sheets Attached. December 22.

IT. 1985. Passaic river sediment study data. International Technology Corporation. In: *Analytical Data Summary Tables Related to Passaic River Study.* Presented to U.S. Environmental Protection Agency Region II, NY and New Jersey Department of Environmental Protection, April 22, 1993.

IT. 1986. Passaic river sediment study data. International Technology Corporation. In: *Analytical Data Summary Tables Related to Passaic River Study.* Presented to U.S. Environmental Protection Agency Region II, NY and New Jersey Department of Environmental Protection, April 22, 1993.

NJDEP. 1985. *A Study of Dioxin (2,3,7,8-Tetrachlorodibenzo-p-Dioxin) Contamination in Select Finfish, Crustaceans, and Sediments of New Jersey Waterways.* New Jersey Department of Environmental Protection, Office of Science and Research, Trenton, NJ. October 30.

NJDEP. 1990. *Polychlorinated Biphenyls (PCBs), Chlordane, and DDTs in Selected Fish and Shellfish from New Jersey Waters, 1986-1987: Results from New Jersey's Toxics in Biota Monitoring Program.* New Jersey Department of Environmental Protection, Trenton, NJ.

NJDEP. 1993. *Polychlorinated Biphenyls (PCBs), Chlordane, and DDTs in Selected Fish and Shellfish from New Jersey Waters, 1988-1991: Results from New Jersey's Toxics in Biota Monitoring Program.* New Jersey Department of Environmental Protection, Trenton, NJ.

OCC. 1994. *Unpublished Data.*

OCC. 1995. *Unpublished Data.*

Suszkowski, D.J. 1978. Sedimentology of Newark Bay, New Jersey: An urban estuarine bay. University of Delaware, Newark, DE.

USACE. 1988. *Data/Analyses Performed from Thirty Sampling Locations*. U.S. Army Corps of Engineers, New York, NY. October.

USACE. 1989. *Passaic River East Bank Stabilization Project, Draft Interpretive Study*. Prepared by U.S Army Corps of Engineers, Waterways Experiment Station, Vicksburg, MI for U.S. Army Corps of Engineers, New York, NY. January.

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3.0 SCREENING-LEVEL HUMAN HEALTH RISK ASSESSMENT

A baseline screening-level human health risk assessment (HHRA) was conducted to evaluate the potential health risks associated with human exposures to chemicals at the Site. The HHRA conforms to the framework established by the National Academy of Sciences (NAS) in 1983 and subsequently adopted by EPA (1987). Consistent with EPA guidance (1987, 1989a), the assessment includes a toxicity assessment (including hazard identification and dose-response assessment), exposure assessment, and risk characterization. As stated in the IWP, and consistent with EPA guidelines (1992), Site-specific data and a number of recent and accepted advances in the science of risk assessment have been incorporated into the HHRA.

3.1 Selection of Chemicals of Potential Concern for Human Health Risk Assessment

The first step in the HHRA is to identify the chemicals of potential concern (CPC). The purpose of identifying CPC is to properly focus the assessment on those chemicals which comprise a significant fraction (>99%) of the theoretical risk. Guidance on the selection of chemicals of potential concern for Superfund sites is presented in the EPA's *Risk Assessment Guidance for Superfund Volume I, Human Health Evaluation Manual (Part A) – Interim Final* (RAGS) (EPA, 1989a). According to RAGS, there are at least four options for selecting the chemicals to be carried through the quantitative risk assessment, once the data quality assessment (see Section 2.2) is complete. The options discussed in detail in RAGS are: 1) group chemicals by class; 2) evaluate frequency of detection; 3) evaluate essential nutrients; and 4) use a concentration-toxicity screen (EPA, 1989a). As described in the IWP, this analysis uses two of these four options to select CPC for the human health risk assessment for the Site: evaluation of essential nutrients and use of a concentration–toxicity screen.

The CPC screening was performed using available chemical data for surface sediments as summarized in Table 2–1. A CPC screening was not performed for chemicals in surface water, as was intended in the IWP, because of the paucity of water quality data collected from the Site, as discussed in Section 2.0. As described below, selection of CPC for the HHRA follows a three-step process: 1) elimination of chemicals that are essential nutrients; 2) initial exclusion of any

chemical which contributes insignificantly to total risk based on a concentration-toxicity screen; and 3) inclusion of potentially bioaccumulative chemicals, including any such chemical initially excluded as a result of the concentration-toxicity screen.

3.1.1 Evaluation of Essential Nutrients

According to EPA (1989a), compounds that are essential human nutrients and are toxic only at very high doses may be eliminated from the quantitative human health risk assessment. As described in the IWP, the following chemicals are considered to meet these criteria and were not retained for the quantitative human health risk assessment: calcium, iron, magnesium, potassium, and sodium.

3.1.2 Initial Sediment Concentration–Toxicity Screen

A sediment concentration–toxicity screen was used to initially screen out chemicals that are unlikely to contribute significantly to the total risk associated with exposure to sediments at the Site. To conduct the screen, "risk factor" scores were calculated for chemicals (other than the essential human nutrients discussed in Section 3.1.1 above) that were detected in the sediments and for which toxicity values could be obtained. The risk factor score is simply the product of the chemical concentration in sediment and the appropriate oral toxicity value. Consistent with EPA guidance (EPA, 1989a), and as discussed further in Section 3.3 (Toxicity Assessment), toxicity values for use in this assessment were obtained from the following sources, in descending order of preference: Integrated Risk Information System (IRIS), Health Effects Assessment Summary Tables (HEAST), EPA criteria documents, and the Environmental Criteria and Assessment Office of the EPA (ECAO) [recently renamed as the National Center for Environmental Assessment (NCEA)]. EPA (1989a) recommends that, if only one exposure route is likely for the medium being evaluated, then the concentration–toxicity screen should employ only toxicity values for that route. Oral exposure to chemicals in sediment (via the food chain) is much more plausible than inhalation or dermal exposure to chemicals in sediments. Therefore, the concentration–toxicity screen was conducted using only toxicity values. Chemicals for which no oral toxicity values were available were retained in the analysis for further evaluation.

Separate risk factors were calculated for carcinogens and for noncarcinogens. For carcinogens, risk factor scores were calculated as follows:

$$R_i = C_i \times CSF_i$$

where,

C_i = 95% upper confidence limit of the arithmetic mean concentration of chemical *i* in sediments; and

CSF_i = oral cancer slope factor for chemical *i*.

For chemicals whose carcinogenicity was assessed using a toxicity equivalency factor (TEF) scheme, the risk factor equation was modified to include the value of the TEF:

$$R_i = C_i \times TEF_i \times CSF_i .$$

Risk factor scores for noncarcinogens were calculated as follows:

$$R_i = C_i / RfD_i$$

where,

RfD_i = oral reference dose for chemical *i*.

The relative risk factor for each chemical was then calculated as the ratio of the individual chemical score to the sum of all (cancer or noncancer) chemical scores:

$$\text{Total Risk Factor} = R_{(tot)} = R_1 + R_2 + R_3 + \ldots + R_n; \text{ and}$$

$$\text{Relative Risk Factor for Chemical } i = R_i/R_{(tot)} .$$

The sediment concentration–toxicity screen is presented in Appendix B. As summarized in Table 3-1, those compounds that contributed less than one percent of the total cancer and noncancer risk factors (either or both, as applicable) were initially eliminated as CPC. Most of the inorganic chemicals from the list of preliminary CPC (Table 2–4), as well as some PAHs, PCBs, and PCDD/Fs were retained as CPC through the concentration-toxicity screen. In addition, those

Table 3-1. Screening for Chemicals of Potential Concern for Human Health Risk Assessment

Chemicals	Percent of Total Risk Factor > 1%	No Toxicity Value Available	Potentially Bioaccumulative Chemical (a)
Inorganics (b)			
Aluminum	X		X
Antimony	X		X
Arsenic	X		X
Barium	X		X
Beryllium	X		X
Cadmium	X		X
Chromium	X		X
Cobalt			X
Copper	X		X
Lead		X	X
Manganese	X		X
Mercury	X		X
Nickel	X		X
Selenium			X
Silver			X
Thallium	X		X
Titanium		X	X
Vanadium	X		X
Zinc			X
Cyanide			
Organics			
PCBs			
TetraCB, 3,3',4,4'- (IUPAC #77)	X		X
PentaCB, 2',3,4,4',5- (IUPAC #123)			X
PentaCB, 2,3',4,4',5- (IUPAC #118)	X		X
PentaCB, 2,3,3',4,4'- (IUPAC #105)	X		X
PentaCB, 2,3,4,4',5- (IUPAC #114)			X
PentaCB, 3,3',4,4',5- (IUPAC #126)	X		X
HexaCB, 2,3',4,4',5,5'- (IUPAC #167)			X
HexaCB, 2,3,3',4,4',5- (IUPAC #156)			X
HexaCB, 2,3,3',4,4',5'- (IUPAC #157)			X
HexaCB, 3,3',4,4',5,5'- (IUPAC #169)			X
HeptaCB, 2,3,3',4,4',5,5'- (IUPAC #189)			X
Aroclor 1248	X		X
Aroclor 1254	X		X
Semivolatiles			
Bis(2-ethylhexyl)phthalate			X
Butyl benzyl phthalate			X
Di-n-butyl phthalate			X
Di-n-octyl phthalate			X
Dichlorobenzene, 1,4-			
Dimethylphthalate		X	
Trichlorobenzene, 1,2,4-			X

Table 3-1. Screening for Chemicals of Potential Concern for Human Health Risk Assessment

Chemicals	Percent of Total Risk Factor > 1%	No Toxicity Value Available	Potentially Bioaccumulative Chemical (a)
PAHs			
Acenaphthene			X
Acenaphthylene			X
Anthracene			X
Benzo(a)anthracene			X
Benzo(a)pyrene	X		X
Benzo(b)fluoranthene	X		X
Benzo(ghi)perylene			X
Benzo(k)fluoranthene			X
Carbazole			
Chrysene			X
Dibenzo(a,h)anthracene	X		X
Dibenzofuran			X
Fluoranthene			X
Fluorene			X
Indeno(1,2,3-c,d)pyrene			X
Methylnaphthalene, 2-		X	X
Naphthalene			
Phenanthrene			X
Pyrene			X
Pesticides			
Aldrin			X
beta-BHC			X
delta-BHC			X
Chlordane			X
alpha-Chlordane	X		X
gamma-Chlordane			X
DDD, 4,4'-			X
DDE, 4,4'-			X
DDT, 4,4'-			X
Dieldrin			X
Endosulfan I			
Endosulfan II			
Endosulfan sulfate			
Endrin			X
Endrin aldehyde			
Endrin ketone			
Heptachlor epoxide (exo)			
Methoxychlor			X

Table 3-1. Screening for Chemicals of Potential Concern for Human Health Risk Assessment

Chemicals	Percent of Total Risk Factor > 1%	No Toxicity Value Available	Potentially Bioaccumulative Chemical (a)
Miscellaneous			
Acetone			
Benzene			
Butanone, 2-			NA
Chlorobenzene			
Chloromethane			
Dichloroethene, 1,2- (total)		X	
Ethyl benzene			
Methylene chloride			
Toluene			
Xylene (total)			
Methylphenol, 4-			NA
Phenol			
TEPH (c)		X	X
Dibutyltin (d)	X		X
Monobutyltin (d)	X		X
PCDD/Fs			
TCDD, 2,3,7,8-	X		X
PeCDD, 1,2,3,7,8-			X
HxCDD, 1,2,3,4,7,8-			X
HxCDD, 1,2,3,6,7,8-			X
HxCDD, 1,2,3,7,8,9-			X
HpCDD, 1,2,3,4,6,7,8-			X
OCDD			X
TCDF, 2,3,7,8-			X
PeCDF, 1,2,3,7,8-			X
PeCDF, 2,3,4,7,8-	X		X
HxCDF, 1,2,3,4,7,8-	X		X
HxCDF, 1,2,3,6,7,8-	X		X
HxCDF, 1,2,3,7,8,9-			X
HxCDF, 2,3,4,6,7,8-			X
HpCDF, 1,2,3,4,6,7,8-	X		X
HpCDF, 1,2,3,4,7,8,9-			X
OCDF			X

a. Organic chemicals with a log Kow > 3.5 were considered to be potentially bioaccumulative (EPA, 1991a).
b. All inorganic chemicals with the exception of cyanide were considered to be potentially bioaccumulative.
c. Total extractable petroleum hydrocarbons (TEPH) are considered to be potentially bioaccumulative, based on the log Kow of PAHs which comprise a significant portion of this group.
d. Organotins are potentially bioaccumulative, similar to other metals.
NA: Kow not available.

chemicals for which there are no oral EPA cancer or noncancer toxicity values reported were retained as CPC for further evaluation in the risk assessment. Chemicals that were not initially retained through the concentration-toxicity screen were primarily volatile organic compounds, as well as the less toxic PAHs, pesticides, PCBs, and PCDD/Fs.

3.1.3 Identification of Chemicals of Potential Concern

The final identification of CPC employs a bioaccumulation screen, and is based on ingestion of aquatic organisms. Because the sediment concentration–toxicity screen above does not consider potential bioaccumulation of compounds in aquatic organisms, and because there are inadequate biological data from the Site to conduct a biota concentration–toxicity screen, those compounds that were initially eliminated based on the sediment concentration–toxicity screen were further evaluated using the bioaccumulation screen. Consistent with EPA guidance (1991a), organic chemicals are considered to be bioaccumulative if their log octanol–water partition coefficient (log K_{ow}) is greater than 3.5. Table 3-1 identifies the organic compounds which are considered bioaccumulative, based on this criterion. Organic compounds that would be eliminated from the assessment based on the initial sediment concentration–toxicity screen, but for which log K_{ow} values of 3.5 or greater were reported, were retained for quantitative assessment of risks from consumption of aquatic organisms. Log K_{ow} values for the preliminary CPC are presented in Section 4.3.

Bioaccumulation screening values, similar to the log K_{ow} are not available for inorganic chemicals. Therefore, to be conservative, all inorganic chemicals with the exception of cyanide were assumed to be potentially bioaccumulative. According to ATSDR (1991), cyanide is not considered bioaccumulative in aquatic organisms. This assumption has been confirmed by the results of a number of studies on chemical concentrations in fish and other aquatic organisms collected from marine and estuarine environments, including the NY/NJ Harbor Estuary (NOAA, 1981, 1990, 1995). Thus, the inorganic chemicals, other than cyanide (and essential nutrients) were retained for the assessment of risks from ingestion of aquatic organisms. The results of the bioaccumulation screen are presented in Appendix B.

Table 3–2 lists the CPC for ingestion of aquatic organisms. All PCDD/Fs, PCBs, and inorganic chemicals, as well as most PAHs, and some pesticides and semivolatile organic compounds were retained as CPC due primarily to their bioaccumulation potential. In addition, those chemicals for

Table 3-2. Chemicals of Potential Concern for the Human Health Risk Assessment (a)

Semivolatiles	Inorganics	Miscellaneous	PAHs	Pesticides	PCBs	PCDD/Fs
Bis(2-ethylhexyl)phthalate	Aluminum	Dichloroethene, 1,2- (total)	Acenaphthene	Aldrin	HeptaCB, 2,3,3',4,4',5,5'- (IUPAC #189)	TCDD, 2,3,7,8-
Butyl benzyl phthalate	Antimony	TEPH	Acenaphthylene	beta-BHC	HexaCB, 2,3,3',4,4',5- (IUPAC #156)	PeCDD, 1,2,3,7,8-
Di-n-butyl phthalate	Arsenic	Dibutyltin	Anthracene	delta-BHC	HexaCB, 2,3,3',4,4',5'- (IUPAC #157)	HxCDD, 1,2,3,4,7,8-
Di-n-octyl phthalate	Barium	Monobutyltin	Benzo(a)anthracene	Chlordane	HexaCB, 2,3',4,4',5,5'- (IUPAC #167)	HxCDD, 1,2,3,6,7,8-
Dimethylphthalate	Beryllium		Benzo(a)pyrene	alpha-Chlordane	HexaCB, 3,3',4,4',5,5'- (IUPAC #169)	HxCDD, 1,2,3,7,8,9-
Trichlorobenzene, 1,2,4-	Cadmium		Benzo(b)fluoranthene	gamma-Chlordane	PentaCB, 2,3',4,4',5- (IUPAC #118)	HpCDD, 1,2,3,4,6,7,8-
	Chromium		Benzo(g,h,i)perylene	DDD, 4,4'-	PentaCB, 3,3',4,4',5- (IUPAC #126)	OCDD
	Cobalt		Benzo(k)fluoranthene	DDE, 4,4'-	PentaCB, 2,3,3',4,4'- (IUPAC #105)	TCDF, 2,3,7,8-
	Copper		Chrysene	DDT, 4,4'-	PentaCB, 2,3,4,4',5- (IUPAC #114)	PeCDF, 1,2,3,7,8-
	Lead		Dibenzo(a,h)anthracene	Dieldrin	PentaCB, 2',3,4,4',5- (IUPAC #123)	PeCDF, 2,3,4,7,8-
	Manganese		Dibenzofuran	Endrin	TetraCB, 3,3',4,4'- (IUPAC #77)	HxCDF, 1,2,3,4,7,8-
	Mercury		Fluoranthene	Methoxychlor	Aroclor 1248	HxCDF, 1,2,3,6,7,8-
	Nickel		Fluorene		Aroclor 1254	HxCDF, 1,2,3,7,8,9-
	Selenium		Indeno(1,2,3-c,d)pyrene			HxCDF, 2,3,4,6,7,8-
	Silver		Methylnaphthalene, 2-			HpCDF, 1,2,3,4,6,7,8-
	Thallium		Phenanthrene			HpCDF, 1,2,3,4,7,8,9-
	Titanium		Pyrene			OCDF
	Vanadium					
	Zinc					

a. Selected based on results of essential nutrient evaluation and bioaccumulation screen. All chemicals without toxicity values were retained.

which there are no EPA cancer or noncancer toxicity values reported were retained as CPC. Chemicals that were not retained were primarily volatile organic and semivolatile organic compounds that are not considered bioaccumulative based on the screening analysis.

3.2 Exposure Assessment

Exposure assessment is the process of measuring or estimating the intensity, frequency, and duration of human or animal exposures to chemicals already present or released into the environment (EPA, 1992; Paustenbach, 1989a; Paustenbach, 1990a). In its most complete form, an exposure assessment should describe the magnitude, duration, schedule, and route of exposure; the size, nature and classes of the human or wildlife populations exposed; and the uncertainties inherent in all estimates (NAS, 1983).

The potential for the occurrence of an adverse health effect associated with exposure to a chemical depends on the degree of systemic uptake (amount absorbed into the blood and tissues). For any route of exposure, the uptake (U) is the product of exposure (E) and the absorption efficiency (A):

$$U = (E)(A).$$

Although a number of different factors are used to quantify exposure, the mathematical relationship shown above holds true for all exposure routes.

EPA (1989a) outlines the following components of an exposure assessment: 1) characterization of exposure setting, including physical setting; 2) identification of potential exposure pathways and potentially exposed populations; and 3) quantification of exposure. The physical and demographic characteristics of the Site that are relevant to the evaluation of potential exposure to CPC are described below. In addition, the models and assumptions used to calculate the pathway-specific uptake of CPC for use in the evaluation of carcinogenic and noncarcinogenic effects are presented.

3.2.1 Characterization of Exposure Setting

The characterization of the exposure setting is based on an evaluation of both the general physical characteristics of the Site and the characteristics of populations potentially exposed to Site-related

chemicals (EPA, 1989a). In Section 3.2.1.1 the physical setting and general conditions of the Site and its historical and current uses are described. In Section 3.2.1.2 the population characteristics that are relevant to the exposure assessment, including demographic information, points of access to the Site, presence of subpopulations of special interest, and behavior patterns which may affect potential exposures are discussed.

3.2.1.1 Physical Setting

During the past century, the tidal Passaic River, including the Site, has been used as a source of industrial process waters, as well as a receiving water for industrial and municipal discharges of wastes from numerous industrial, commercial, and transportation facilities, and domestic sources. The historical and current mass loadings of hazardous chemicals to the Site are associated with several ongoing sources including, but not limited to, POTWs and CSOs, industrial waste discharged either directly to the estuary or through POTWs, stormwater runoff, and accidental spills of petroleum products and hazardous chemicals.

The vast majority of the shoreline adjacent to the Site consists of operating and/or abandoned industrial properties. Shorelines are characterized by the presence of wooden and stone bulkheads, riprap, parking lots, highways, and railway lines. Although there are scattered and limited vegetated areas along the shoreline of the Site, these locations are typically very narrow areas between the river and adjacent highways or industrial facilities. The physical characteristics of the Site are described more fully in Appendix E.

As described in detail in Section 1.0, human activity at the Site during the past two centuries has resulted in severe adverse impacts to natural resources and aesthetic qualities of the Site. These impacts have included reduced biological diversity and abundance and reduced opportunities for recreational activities due to limitations on access, and effects on water quality. As a result, there has been decreased use of the Site for fishing and recreational purposes (NJMSC, 1987; Pearce et al., 1988); and no significant commercial fishery has operated within the Passaic River since the early 1900s (McCormick and Quinn, 1975; Crawford et al., 1994). Since the mid-1800's, there has been a concern for potential adverse human health effects associated with the degraded condition of the Site watershed (Brydon, 1974). Recognition of the health risks associated with pathogenic contamination from sewage loadings to the Site has resulted in a prohibition on

shellfish harvesting since 1970. As a result of the long-standing water quality concerns and extensive historic and present use of the Site as an industrial area, it is unlikely that any substantial change in land use could occur in the near future.

3.2.1.2 Identification of Potential Exposure Pathways and Potentially Exposed Populations

In a human health risk assessment, potentially exposed populations are identified for quantitative evaluation, based on the likely uses of the Site and the types of people that may frequent its use. As discussed in Section 3.2.1.1, the Site has not been used either for commercial fishing or shellfishing since the early 1900s. In addition, because of saline water conditions, the Site has never been used as a potable water supply.

Recreational activities, including swimming, boating, fishing and/or crabbing, are the primary means by which a population could be expected to be exposed to contaminated media in this estuarine environment. Recreational use of the Site, however, is minimal for several reasons. First and foremost, access to the river at the Site is extremely limited by the presence of numerous industrial and commercial facilities, railroad tracks, and highways lining the shores. In fact, based on the shoreline (habitat) survey conducted by ChemRisk in August 1994 (see Appendix E), public access to the Site for recreational purposes is limited to an approximate 100-foot portion of the right bank in River Bank Park in the Arlington Reach. Other possible points of access to the Site for fishing or crabbing are limited to a few scattered locations including parking lots, vacant lots, bridges, and a single boat launch site.

Swimming is also unlikely to occur at the Site. As noted above, public access to the River, and its shorelines is extremely limited, being comprised primarily of bulkhead and rip-rap and areas too steep or too rocky to provide swimmers with suitable access. The river bottom is not sandy, but consists primarily of mud. Furthermore, the poor water quality and aesthetic conditions of the Site are well-known, and for these reasons, it is unlikely that individuals would choose to swim or wade at the Site. This has been confirmed by the National Oceanic and Atmospheric Administration's (NOAA) Office of Marine Pollution Assessment which stated that "much of the ocean, harbor, and river frontage (of the Hudson-Raritan Estuary including the lower Passaic River) that is not topographically definable as 'beach' is officially unavailable for bathing" (NOAA, 1981). In addition, in its State Water Quality Inventory Report, the New Jersey Department of

Environmental Protection (NJDEP) stated that the Lower Passaic River "will not support the primary contact (swimmable) designated use" (NJDEP, 1992); in other words, water quality has been historically degraded to the point that swimming is not a viable use for the Site. Residents of the Newark, NJ area who are interested in swimming or wading have ready access to a variety of nearby alternative recreational areas; numerous public swimming pools and beaches along the New Jersey shore provide more sanitary and aesthetically superior swimming locales. For example, the Upper Passaic River (above Dundee Dam) and a number of freshwater lakes, reservoirs, and rivers within the Passaic River watershed (Hauge et al., 1990) provide a scenic alternative to recreation in proximity to the Site. Given the access limitations, aesthetic deterrence, and availability of more suitable areas, it is highly unlikely that anyone would choose to swim or wade at the Site. Consequently, potential exposure through direct dermal contact with contaminated water or sediments by residents or visitors engaging in recreational uses of the Site were not quantitatively evaluated in this assessment.

Due to the degraded conditions and low biological diversity of the Site, as well as the presence of substantial high quality fisheries in northern New Jersey, it is unlikely that most anglers would choose to recreationally fish or crab at the Site. In addition, there are numerous well-posted regulatory fishing bans in the River that have been instituted. However, for the purposes of this screening-level analysis, it will be assumed that some consumption of fish and crabs from the Site does occur. This is in spite of the existing regulatory fishing bans in the River. Therefore, consumption of fish and crabs from the Site by urban anglers are evaluated in this assessment. To appropriately characterize potential exposures of those recreational anglers, studies of urban angler populations and their behavior pattern were evaluated.

Behavior Patterns of Urban Angler Populations

Belton et al. (1985) conducted a creel survey of urban recreational anglers frequenting the Hudson River, Upper New York Bay, and Newark Bay areas. In this creel survey, researchers from Rutgers University interviewed anglers at six shoreline sites chosen from among 11 access locations along the upper New York Bay, Kill Van Kull, Newark Bay, and Lower Hudson River. Using a questionnaire format, the investigators obtained information on race/ethnicity, age, gender, size of home living group, location of residence, frequency of fishing, disposition of the catch,

quantity consumed, and method of cooking (if consumed). Additional questioning sought to characterize the anglers' awareness of fishing advisories and perceptions of risks associated with consumption.

Results of the survey indicated that the typical angler in the area was white (86%), greater than 60 years of age (59%), who fished frequently (46% weekly) as a source of food (59%). In addition, although quantitative data regarding gender were not reported, Belton et al. (1985) indicated that the majority of anglers were male. Table 3-3 presents the demographic makeup of the surveyed population and the results of the creel survey as reported by Belton et al. (1985). Forty-four percent (44%) of those surveyed indicated that they would either consume or give away their catch; the remaining 55% planned to use the fish for non-food purposes. Frequency of fishing was reported to range from daily to once per month; it is important, however, to recognize that creel surveys tend to oversample frequent anglers and thus are likely to overestimate the distribution of fishing frequencies and/or consumption rates among the entire angling population (Puffer et al., 1981; Price et al., 1994; Ebert et al., 1994). Based on the available data, it appears that the survey by Belton et al. (1985) represents the best source of information regarding the likely characteristics of the population of anglers who might fish at the Site.

Because the Belton et al. (1985) survey is a decade old, some changes in the demographic makeup and/or fishing preferences of the population might be expected. Significant shifts may have occurred if external pressures exist. Pressures that may impact the angling population are public awareness of the water quality and existing fishing bans, as well as changes in economic conditions. For example, the poor water quality at the Site has been increasingly publicized since 1985. Therefore, it is likely that perceptions of risk among the anglers have increased, thereby reducing either the number of anglers willing to consume their catch or the frequency of fishing in these areas (NJDEP, 1995). Further, with specific respect to the Site, a complete prohibition on the sale or consumption of fish from the lower Passaic River (below Dundee Dam) has existed since 1983 (NJ Administrative Order No. EO40-19). To the extent that this fishing prohibition has been adequately publicized, it likely serves as a serious deterrent to individuals considering fishing at the Site or consuming their catch. Alternatively, the increased publicity of water quality concerns and fishing prohibitions may have shifted the demographic makeup of the angling

Table 3-3. Demographics and Fishing Patterns Reported by Belton et al. (1985)

	Belton et al. (1985) Newark-New York Bays
Gender	(a)
Race	
Caucasian	86
Black	7
Hispanic	6
Asian	1
Other	
Age	
5 - 19	7 (b)
>20	93
Fishing Frequency (c)	
Daily	21
Weekly	65
Monthly	13
< Monthly	-
Disposition of Catch	
Eat	21
Give Away	23
Other	55

* Some categories may not sum to 100 due to rounding or inclusion of additional/fewer response options.
a. Quantitative data regarding gender not presented
b. Percent of fishermen
c. Daily considered >3x/week; weekly was 1-3x/week; monthly was 1-3x/month; <monthly was <1x/month

population. However, in the absence of more recent data in a comparable location, it appears reasonable to assume that the potentially exposed population for the Site resembles the angling population surveyed by Belton et al. (1985).

In summary, this screening-level HHRA defines the most likely exposed population as that group of urban resident anglers (and their families) who are unable or unwilling to travel to more desirable fishing locales. Some proportion of this population will likely practice catch-and-release fishing (i.e. not consume their catch) and, thus, will not be exposed to contaminants taken up by fish. However, in the absence of Site-specific data, it will be assumed that some fish and crabs taken from the Site portion of the River are consumed.

Subpopulations of Potential Concern

EPA (1989a) defines subpopulations of potential concern as those subgroups which are at increased risk from chemical exposures as a result of increased sensitivity, unusually high exposure potential, or exposure from other sources. Among those individuals potentially exposed to Site contaminants, subgroups that potentially may be at increased risk of health effects include women of childbearing age and children. Women of childbearing age may potentially incur an increased risk of reproductive effects or, if pregnant, their offspring may potentially incur an increased risk of developmental effects. Children may face an increased health risk a result of lower body weight and underdeveloped physiological systems.

It is not possible to quantify, from a toxicological perspective, the increased risk experienced by these subpopulations of potential concern. Although some toxicity values adopted by EPA (reference doses, specifically) may be derived to protect against reproductive or developmental effects, the vast majority are associated with systemic critical endpoints. It is important to note that, in the derivation of reference doses, EPA includes additional uncertainty factors to account for increased sensitivity in the population and for the absence of information on reproductive and developmental effects (EPA, 1989a). Therefore, an added level of conservatism is included in the derivation of most toxicity values in order to account for the possible existence of sensitive subpopulations. In the absence of data to quantify the susceptibility of sensitive subpopulations,

EPA (1992) recommends that the risks for these individuals be treated as part of the variability in the general population. Thus, in this assessment, subpopulations of potential concern will be considered within the variability of the general population.

Based on the demographic information provided by Belton et al. (1985), the angling population is unlikely to contain a significant proportion of women (of any age) or children. Although anglers may share their catch with family members, women are likely to consume at approximately the same rate per body weight as men, and children are known to consume less fish than adults (Rupp, 1980).

The "subsistence fisherman" is another potentially sensitive subpopulation that is sometimes considered in risk assessments involving the consumption of fish. As noted in the EPA's (1989a) RAGS, the existence of subsistence fishing should be quantitatively evaluated in the assessment when there is clear evidence that such fishing occurs (e.g., Native Americans harvesting salmon from the Columbia River). However, it is implausible to expect, given the Site conditions described above, that subsistence fishing occurs at the Site. This is supported by the fact that, of the numerous published accounts of fishing habits in and around the Site (Belton et al., 1983, 1985), there has never been a single reported incident of one or more persons accessing the River for the purpose of subsistence fishing. Furthermore, the consumption rate estimates derived for this assessment were taken from creel surveys that included both recreational and subsistence fishermen. Therefore, subsistence fishing is not considered as a separate pathway in this assessment.

3.3 Quantification of Exposure

As discussed above, consumption of fish and shellfish is the only plausible pathway of human exposure to Site-related chemicals. In this section the exposure parameters used to estimate chemical uptake via ingestion of fish or shellfish are described. Chemical uptake, or dose, is expressed in units of milligram of chemical per kilogram of body weight per day (mg/kg-day), and is calculated using the following general equation:

$$\text{Intake} = \text{CF} \times \text{CR} \times \text{A} \times \text{EF} \times \text{ED} \times 1/\text{BW} \times 1/\text{AT}$$

where,

CF = Chemical concentration in fish (mg/kg);
CR = Fish consumption rate (grams/day);
A = The absorption factor (unitless);
EF = The exposure frequency or rate of incidence of exposure (days/year);
ED = Length of exposure (years);
BW = The body weight over the exposure duration (kg); and
AT = Averaging time (days).

The parameters used to calculate intake for characterization of the carcinogenic and noncarcinogenic risks are described below. The uncertainties associated with the assumptions used in the exposure assessment are discussed in Section 3.5.3 (Identification of Uncertainties).

3.3.1. Exposure Point Concentrations

Consistent with the IWP, concentrations of organic chemicals in striped bass and blue crab were estimated from the 95% UCL of the arithmetic mean of the Site sediment data using a food web model, as described in Section 4.4.2.1. The model estimated both lipid normalized (µg/kg-L) and wet weight concentrations of organic chemicals (µg/kg) in striped bass and blue crab. Concentrations of inorganic chemicals in striped bass and blue crab were estimated using empirical relationships derived from the scientific literature as discussed in Section 4.4.2.1.

For both organic and inorganic chemicals in blue crab and striped bass, the edible tissue concentration was used to represent the exposure point concentration, since the vast majority of anglers consume only the muscle tissue (i.e., backfin and claw muscle) of crab and the fillets of fish (Landolt et al, 1985; EPA, 1989b; Ebert et al., 1994). For blue crab, chemical concentrations in the edible muscle tissue were used as exposure point concentrations. For organic chemicals, the concentration in muscle was calculated by multiplying the lipid normalized chemical concentration in whole crab by the mean percent of lipid in the muscle tissue of crab (0.78 %) (Belton et al., 1985; Hauge et al., 1990, 1993) to derive a muscle concentration (mg/kg). Similarly, for striped bass, chemical concentrations in edible fillets were used as the exposure point concentrations. For

organic chemicals, the concentration in the fillet was calculated by multiplying the lipid normalized chemical concentration in whole fish by the mean percent of lipid in striped bass fillets (2.28%) (Belton et al., 1985; Hauge et al., 1990, 1993) to derive a fillet tissue concentration (mg/kg).

For inorganic chemicals, the exposure point concentrations in striped bass were calculated by multiplying the estimated whole body tissue concentration by 0.3; this conservatively assumes that 30% of the metal concentrations in striped bass are available in the fillet, since fillets comprise about 30 percent of the mass of a fish (EPA, 1989b; Ebert et al., 1994). Similarly, the exposure point concentrations for inorganic chemicals in blue crab were calculated by multiplying the whole body concentration by 0.3. This is a conservative assumption, since the edible muscle tissue (i.e., backfin and claw muscle) of crab likely comprises much less than 30 percent of the whole body mass.

Exposure point concentrations for striped bass and blue crab are presented in Table 3-4. For ingestion of striped bass fillets, humans are hypothetically exposed to a wide range of chemicals including PCDD/Fs (3.4 x 10^{-8} to 9.8 x 10^{-6} mg/kg), PAHs (9.3 x 10^{-7} to 1.1 x 10^{-5} mg/kg), coplanar PCBs (1.6 x 10^{-7} to 0.00068 mg/kg), PCB Aroclor mixtures (0.0036 to 0.015 mg/kg), pesticides (5.0 x 10^{-5} to 0.00093 mg/kg), inorganics (0.047 to 1,100 mg/kg), and semivolatiles (2.0 x 10^{-6} to 0.00011 mg/kg). Similarly, for ingestion of blue crab (whole body), humans are hypothetically exposed to PCDD/Fs (2.6 x 10^{-7} to 0.00011 mg/kg), PAHs (0.00014 to 0.0012 mg/kg), coplanars PCBs (1.2 x 10^{-6} to 0.0046 mg/kg), PCB Aroclor mixtures (0.024 to 0.10 mg/kg), pesticides (0.00040 to 0.018 mg/kg), inorganics (0.095 to 2,200 mg/kg) and semivolatiles (8.6x10^{-5} to 0.0055 mg/kg). The exposure point concentrations in Table 3-4 were used to calculate both typical and reasonable maximum intakes for all CPC for use in the carcinogenic and noncarcinogenic HHRA.

3.3.2 Exposure Parameters

Fish Consumption Rate

The amount of fish consumed by a population of anglers varies, depending upon the numbers and types of waterbodies fished and the characteristics of the angler population. Fish consumption also depends on factors such as climate, fish species present, fish productivity, waterbody access, and the size of the angler population. Historically, fish consumption estimates ranging from 1.2 to 180

Table 3-4. Exposure Point Concentrations for Humans Consuming Blue Crab and Striped Bass

Chemicals of Potential Concern	Exposure Point Concentrations (mg/kg)	
	Blue Crab	*Striped Bass*
PCDD/Fs		
TCDD, 2,3,7,8-	$1.4x10^{-5}$	$2.1x10^{-6}$
PECDD, 1,2,3,7,8-	$2.6x10^{-7}$	$3.6x10^{-8}$
HxCDD, 1,2,3,4,7,8-	$2.6x10^{-7}$	$3.4x10^{-8}$
HxCDD, 1,2,3,6,7,8-	$7.8x10^{-7}$	$1.0x10^{-7}$
HxCDD, 1,2,3,7,8,9-	$3.9x10^{-7}$	$5.0x10^{-8}$
HpCDD, 1,2,3,4,6,7,8-	$9.4x10^{-6}$	$1.0x10^{-6}$
OCDD	0.00011	$9.8x10^{-6}$
TCDF, 2,3,7,8-	$3.2x10^{-6}$	$5.0x10^{-7}$
PECDF, 1,2,3,7,8-	$2.3x10^{-6}$	$3.6x10^{-7}$
PECDF, 2,3,4,7,8-	$4.4x10^{-6}$	$6.6x10^{-7}$
HxCDF, 1,2,3,4,7,8-	$3.3x10^{-5}$	$4.6x10^{-6}$
HxCDF, 1,2,3,6,7,8-	$5.1x10^{-6}$	$7.1x10^{-7}$
HxCDF, 1,2,3,7,8,9-	$5.9x10^{-7}$	$8.2x10^{-8}$
HxCDF, 2,3,4,6,7,8-	$1.8x10^{-6}$	$2.5x10^{-7}$
HpCDF, 1,2,3,4,6,7,8-	$7.2x10^{-5}$	$8.4x10^{-6}$
HpCDF, 1,2,3,4,7,8,9-	$1.7x10^{-6}$	$2.1x10^{-7}$
OCDF	$7.6x10^{-5}$	$5.9x10^{-6}$
PAHs		
Acenaphthene	0.00027	$2.1x10^{-6}$
Acenaphthylene	0.00020	$1.6x10^{-6}$
Anthracene	0.00033	$3.2x10^{-6}$
Benzo(a)anthracene	0.00059	$9.8x10^{-6}$
Benzo(a)pyrene	0.00059	$9.3x10^{-6}$
Benzo(b)fluoranthene	0.00059	$9.3x10^{-6}$
Benzo(ghi)perylene	0.00029	$2.7x10^{-6}$
Benzo(k)fluoranthene	0.00059	$9.3x10^{-6}$
Chrysene	0.00067	$1.1x10^{-5}$
Dibenzo(a,h)anthracene	0.00014	$9.3x10^{-7}$
Dibenzofuran	0.00023	$1.8x10^{-6}$
Fluoranthene	0.0012	$2.0x10^{-5}$
Fluorene	0.00026	$2.1x10^{-6}$
Indeno(1,2,3-c,d)pyrene	0.00031	$3.0x10^{-6}$
Methylnaphthalene, 2-	0.00025	$2.0x10^{-6}$
Phenanthrene	0.00078	$7.8x10^{-6}$
Pyrene	0.0012	$1.8x10^{-5}$
PCBs and PCB Coplanars		
Aroclor 1248	0.10	0.015
Aroclor 1254	0.024	0.0036
TetraCB, 3,3',4,4'- (IUPAC #77)	0.00067	0.00010
PentaCB, 2',3,4,4',5- (IUPAC #123)	0.00044	$6.8x10^{-5}$
PentaCB, 2,3',4,4',5- (IUPAC #118)	0.0046	0.00068
PentaCB, 2,3,3',4,4'- (IUPAC #105)	0.0023	0.00035
PentaCB, 2,3,4,4',5- (IUPAC #114)	0.00014	$2.1x10^{-5}$
PentaCB, 3,3',4,4',5- (IUPAC #126)	$2.7x10^{-5}$	$4.1x10^{-6}$
HexaCB, 2,3',4,4',5,5'- (IUPAC #167)	0.00049	$7.1x10^{-5}$
HexaCB, 2,3,3',4,4',5'- (IUPAC #157)	0.00010	$1.5x10^{-5}$
HexaCB, 2,3,3',4,4',5- (IUPAC #156)	0.00034	$5.0x10^{-5}$
HexaCB, 3,3',4,4',5,5'- (IUPAC #169)	$1.2x10^{-6}$	$1.6x10^{-7}$
HeptaCB, 2,3,3',4,4',5,5'- (IUPAC #189)	$9.4x10^{-5}$	$1.2x10^{-5}$

Table 3-4. Exposure Point Concentrations for Humans Consuming Blue Crab and Striped Bass

Chemicals of Potential Concern	Exposure Point Concentrations (mg/kg)	
	Blue Crab	*Striped Bass*
Pesticides		
Aldrin	0.0013	0.00020
Alpha-Chlordane	0.0025	0.00039
Beta-BHC	0.00040	5.0×10^{-5}
DDD, 4,4'-	0.018	0.0027
DDE, 4,4'-	0.0046	0.00071
DDT, 4,4'-	0.0054	0.00082
Delta-BHC	0.00046	6.2×10^{-5}
Dieldrin	0.00096	0.00010
Endrin	0.0034	0.00050
Gamma-Chlordane	0.0029	0.00046
Methoxychlor	0.0062	0.00093
Inorganics		
Aluminum	2,200	1,100
Antimony	1.5	0.75
Arsenic	2.3	1.1
Barium	35	17
Beryllium	0.19	0.092
Cadmium	1.1	0.54
Chromium	1.1	0.54
Cobalt	2.3	1.1
Copper	39	20
Lead	12	6.0
Manganese	65	32
Mercury	0.59	0.29
Nickel	9.8	4.9
Selenium	0.24	0.12
Silver	1.1	0.53
Thallium	0.095	0.047
Titanium	74	37
Vanadium	6.5	3.2
Zinc	94	47
Semivolatiles		
Bis(2-Ethylhexyl)phthalate	0.0055	8.2×10^{-5}
Butyl benzyl phthalate	0.00020	3.4×10^{-6}
Di-n-butyl phthalate	0.00022	3.6×10^{-6}
Di-n-octyl phthalate	0.00027	4.8×10^{-6}
Dimethylphthalate	8.6×10^{-5}	0.00011
Trichlorobenzene, 1,2,4-	0.00023	2.0×10^{-6}
Miscellaneous		
Dichloroethene, 1,2- (total)	1.8×10^{-6}	6.6×10^{-7}
Dibutyltin	0.050	0.025
Monobutyltin	0.071	0.071

g/day have been used or recommended for use by EPA in risk assessments and regulatory proceedings (EPA, 1986, 1989a, 1989b, 1989c, 1991a,b, 1992, 1993a). The differences in these consumption rates reflect variations in waterbody type, target population, fishery type, region, and study methodology. All of these factors must be considered in evaluating possible fish consumption rates for use in the estimation of risks from consumption of fish from the Site.

Ideally, rates of fish consumption used to estimate exposure to a particular waterbody would be derived from site–specific information on local consumption patterns. When this information is not available, and consumption rates must be estimated based on data from other areas, it is critical that those data that best simulate the situation to be assessed are selected for deriving quantitative fish consumption rates.

Specifically, in selecting a fish consumption rate to be used in estimating fish intake from a specific waterbody like the tidal Passaic River, it is important that the fish consumption rate be derived from a study or studies that are representative of the Site with respect to the type of waterbody and target population being evaluated. For example, marine and estuarine fish consumption estimates should not be based on studies of freshwater fisheries because there are likely to be differences between the species present, the relative productivities of the waters, and the preferences of the fish consumers. Likewise, if the consumption of fish from a single waterbody is being evaluated, it is most appropriate to base the rate of intake on a study of intake from a similar, individual waterbody. If there are no commercial fisheries on the waterbody of interest, as is the case for the Site, then the rates of intake used should be based on studies which considered only the intake of self-caught fish, because recreational fishing is the only potential source of fish from the waterbody. In this situation, consumption estimates should not be based on studies that have considered consumption of fish obtained from restaurants, markets, or other non-recreational angling sources in their estimated rates.

A second consideration in determining the appropriateness of a given fish consumption survey is the survey method used to obtain data. There are a variety of different survey methods which have been used to collect data on consumption rates; the most common methods are creel surveys and recall surveys. Typically, creel surveys are used to evaluate angler effort and harvest rates for a

single point in time on a particular waterbody, while recall surveys generally collect longer term information and may collect data on a number of waterbodies within a given area (Ebert et al., 1994).

Because creel surveys involve on-site interviews during the course of a fishing season, they tend to be biased toward the more frequent angler. This occurs because the probability of encountering a frequent angler is much greater than the probability of encountering a less frequent angler (Puffer et al., 1981; Price et al., 1994; Ebert et al., 1994). Thus, because frequent anglers are oversampled, consumption estimates based on creel surveys are likely to be more representative of frequent anglers and may not be representative of the total population of anglers using an individual waterbody.

Recall surveys usually target entire populations of anglers through, for example, mass mailings to fishing license holders. In this manner, recall surveys are more likely to be representative of the entire angling population than creel surveys. Recall surveys typically request that individuals summarize their fishing activities over the course of a season or a year. Because recall surveys do not require the extrapolation from short-term measurements to annual rates, the effect of short-term variability is minimized. Long-term recall surveys may be subject to recall bias if individuals systematically over- or under-estimate their fishing patterns over long time periods (Ebert et al., 1994).

As previously discussed, for a number of reasons the population at risk from the consumption of contaminated fish and crabs from the Site probably comprises a very small fraction of the general population from the Newark metropolitan region. Most importantly, because nearly the entire shoreline of the Site is comprised of private industrial/commercial properties, there is little public access to the Site from shore that affords anglers an opportunity to fish or crab. This restricts fishing access to the Site primarily to recreational boaters. Given the poor aesthetics, impacted water quality, and low biological diversity of the Site, as compared to many other regional waterways, it is unlikely that anglers who own boats would choose the Site as a fishing area.

Information on fish consumption rates among urban recreational anglers was obtained from the scientific literature, and from EPA guidance documents (EPA, 1989b,c). A literature search was performed to identify recent publications on fishing and fish consumption rates. Ebert et al. (1994)

present a framework for selecting fish consumption rates for exposure assessment in the absence of site–specific survey data. In their review of available fish consumption data, Ebert et al. (1994) identified three studies reporting consumption patterns for sport-caught marine fish among urban anglers: Pierce et al. (1981), Puffer et al. (1981), and Landolt et al. (1985, 1987). However, because Landolt et al. (1985, 1987) did not provide annual estimates of fish consumption, the data from that survey are considered of limited value for risk assessment. Price et al. (1994) published a reanalysis of the Puffer et al. (1981) and Pierce et al. (1981) surveys, addressing sampling bias in the original studies. Brief summaries of these studies are presented below.

Pierce et al. (1981)

Pierce et al. (1981) surveyed anglers for four or five days during the summer and fall seasons of 1980, conducting interviews with anglers at 5 locations on Commencement Bay in Puget Sound, Washington. Interviews were only conducted with anglers who had creeled fish that day. Interviewees were asked to provide information on fishing frequency, disposition of catch (consumption, bait, release), size of home living group, and place of residence. In addition, researchers recorded the angler's approximate age, gender, race, mode of fishing, number of fish caught, and average weight of fish caught. While Pierce et al. (1981) did not estimate consumption rates for the anglers, EPA (1989c) estimated a distribution of fish consumption rates based on the data provided by Pierce et al. (1981). EPA (1989c) estimated median and 90th percentile consumption rates of 23 and 54 g/day based on the data of Pierce et al. (1981).

Puffer et al. (1981)

Puffer et al. (1981) investigated the fish consumption habits of marine fishermen at 12 fishing sites along coastal Los Angeles Bay. Interviews were conducted approximately 3 times per month at each site, on different days of the week and at different times of day, for one year. Although all of the fishermen observed in the study were counted, only those fishermen who had creeled fish were subsequently interviewed. The authors reported that the median consumption rate for those successful anglers was 37 g/day; the 90th percentile was reported to be 225 g/day.

Price et al. (1994)

Price et al. (1994) published a reanalysis of the surveys of Pierce et al. (1981) and Puffer et al. (1981) that corrected for the bias inherent in the creel survey sampling design. The authors demonstrated that creel surveys are strongly biased toward more frequent anglers because frequent

anglers are more likely to be present when interviewing occurs than infrequent anglers. Due to this bias, the median fish intake for the survey population is substantially higher than the median consumption rate for the total population of anglers using the body of water. Price et al. (1994) corrected for this bias, deriving median intake rates (for the angling population) of 2.9 and 1.0 g/day for Puffer et al. (1981) and Pierce et al. (1981), respectively. The authors also provided estimates of the 90th percentile consumption rates for the total angling populations in these two studies of 35 and 13 g/day, respectively.

Historically, EPA (1989c) has recommended that the data from Puffer et al. (1981) and Pierce et al. (1981) be used to represent consumption rates for recreational angling from a large waterbody with widespread contamination. However, for this assessment, an average of the consumption rates recently calculated by Price et al. (1994), based on the reanalysis of the Puffer et al. (1981) and Pierce et al. (1981) data, represent a better approximation of the consumption of fish and shellfish from the Site. Because the consumption rates calculated by Price et al. (1994) correct for the inherent bias in the creel survey design used by both Puffer et al. (1981) and Pierce et al. (1981), while the original consumption rates do not, the Price et al. (1994) estimates more appropriately represent consumption by the total population of anglers from an estuarine waterway.

The consumption rates calculated by Price et al. (1994) likely overestimate consumption of fish from the Site for several reasons. First, the Puffer et al. (1981) and Pierce et al. (1981) studies, on which the Price et al. (1994) calculation is based, evaluated consumption rates for much larger and more desirable fishing areas than the Site. Neither Los Angeles Harbor nor Commencement Bay, respectively, were subject to fishing prohibitions at the time of the surveys; thus, fishing in those areas was not restricted. In addition, both surveys were conducted in areas in which there was not the widespread knowledge of water quality concerns that exist at the Site. Furthermore, the areas surveyed in both studies were readily accessible to the public for recreational fishing, in contrast to the extremely limited access available at the Site. Finally, the Site, unlike west coast estuaries, such as Los Angeles Harbor or Commencement Bay, is subject to seasonal restrictions on recreational fishing due to : 1) inclement weather conditions from the late fall to early spring; and 2) the seasonal availability of migratory fish and crabs which comprise the limited number of species that may be present at the Site. For these organisms, particularly striped bass and blue crab, it would be conservative to assume that the residence time is 6 months of the year. In short, it is plausible to conclude that the refined analysis of Price et al. (1994) overestimates consumption

rates at the Site by at least 2-fold. This is consistent with a position paper recently submitted to EPA Region II which supports the use of 50% of the Price et al. (1994) fish consumption rates for the Upper Hudson River (ChemRisk, 1994). In this analysis, it was noted that, due to the relatively severe weather conditions of the Upper Hudson River versus those of Los Angeles Harbor and Commencement Bay, a 50% seasonal correction factor should be applied to the Price et al. (1994) consumption rates.

The consumption rates used in this assessment to represent typical and reasonable maximum exposures (RME) are 1 g/day and 12 g/day, respectively. These respective values represent the averages of the 50th and 90th percentile consumption rates (for total fish and shellfish consumption) calculated by Price et al. (1994) based on a reanalysis of the data of Puffer et al. (1981) and Pierce et al. (1981) and incorporation of a 50% seasonal correction factor.

To estimate exposures specifically to finfish and shellfish, these total consumption rates were allocated to these two groupings based on data compiled by Javitz (1980) and reported by EPA (1989b). Javitz (1980) reported mean species-specific fish consumption rates for fish consumers in the United States based on responses to a 1973-1974 survey conducted by NPD Research, Inc. The proportion of total fish consumption represented by finfish or shellfish was calculated based on these data. The total fish/shellfish consumption rate was first calculated. Then, the ratio of finfish consumption to total fish consumption was estimated to be 0.78; the remaining fish consumption was assumed to be comprised of shellfish (0.22). These proportions were used to allocate the total fish consumption rates from Price et al. (1994) between finfish and shellfish. Because striped bass and blue crab are two commercially/recreationally valuable species that are present in the tidal Passaic River (see discussion in Section 4.1), these organisms were used to represent total finfish consumption and total shellfish consumption respectively. The consumption rates for striped bass were estimated to be 0.78 g/day (typical) and 9.4 g/day (RME). For blue crab, the consumption rates were estimated to be 0.22 g/day for typical and 2.6 g/day for the RME.

Absorption Fraction

Consistent with EPA (1989b) guidance, an absorption coefficient of 1.0 was used in the calculation of intake. Use of an absorption fraction of 1.0 assumes that the human absorption efficiency of CPC from ingestion of fish and shellfish is equal to that of the laboratory animal in the study upon which the cancer slope factor or reference dose is based (EPA, 1989b).

Exposure Frequency

An exposure frequency of 365 days was used in the intake calculations because the fish consumption rates are annualized. Therefore, in order to be consistent with the fish consumption rate, it was assumed that people consume fish from the Site everyday.

Exposure Duration

As recommended by EPA (1991a) for reasonable maximum exposure residential scenarios, a 30-year exposure duration was applied in this assessment. Use of this assumption implies that a resident fishes from the Site portion of the Passaic River throughout a 30-year period. For the typical case, an exposure duration of 9 years was utilized; this value represents the 50th percentile for the number of years spent at a single residence (EPA, 1989c).

Body Weight

The EPA (1989c) recommends 70 kg as an appropriate estimate of body weight for adults. The value was derived from mean adult male and mean adult female body weights (EPA, 1989c).

Averaging Time

For carcinogens, intakes were calculated by averaging the dose over a lifetime of 70 years or 25,550 days (EPA, 1989a). For the evaluation of noncarcinogenic effects, the exposure duration of 10,950 days (30 years) was used as the averaging time for the reasonable maximum exposure. For the typical exposure case, an averaging time of 3,285 days (9 years) was used (EPA, 1989a).

3.3.3 Estimation of Chemical Intakes

Using the estimated exposure point concentrations presented in Table 3-4, and the exposure parameters described above, average daily intakes (ADIs) and lifetime average daily intakes (LADIs) resulting from the consumption of striped bass and blue crab were calculated for CPC; these are presented in Tables 3-5 and 3-6, respectively. Appropriate toxicity values were applied to ADIs and LADIs (cancer slope factors (CSFs) and TEFs, where applicable, for carcinogenic effects; reference doses (RfDs) for noncarcinogenic effects) in Section 3.5 (Risk Characterization) to assess the potential risk to human health associated with exposure to CPC.

Table 3-5. Lifetime Average Daily Intake (Carcinogenic) Estimates (mg/kg-day) (a)

Chemicals of Potential Concern	Blue Crab		Striped Bass	
	Typical	RME (b)	Typical	RME (b)
PCDD/Fs				
TCDD, 2,3,7,8-	$5.7x10^{-12}$	$2.2x10^{-10}$	$3.0x10^{-12}$	$1.2x10^{-10}$
PeCDD, 1,2,3,7,8-	$5.2x10^{-14}$	$2.0x10^{-12}$	$2.6x10^{-14}$	$1.0x10^{-12}$
HxCDD, 1,2,3,4,7,8-	$1.0x10^{-14}$	$4.1x10^{-13}$	$4.9x10^{-15}$	$2.0x10^{-13}$
HxCDD, 1,2,3,6,7,8-	$3.2x10^{-14}$	$1.2x10^{-12}$	$1.5x10^{-14}$	$6.0x10^{-13}$
HxCDD, 1,2,3,7,8,9-	$1.6x10^{-14}$	$6.2x10^{-13}$	$7.2x10^{-15}$	$2.9x10^{-13}$
HpCDD, 1,2,3,4,6,7,8-	$3.8x10^{-14}$	$1.5x10^{-12}$	$1.4x10^{-14}$	$5.8x10^{-13}$
OCDD	$4.4x10^{-14}$	$1.7x10^{-12}$	$1.4x10^{-14}$	$5.6x10^{-13}$
TCDF, 2,3,7,8-	$1.3x10^{-13}$	$5.1x10^{-12}$	$7.2x10^{-14}$	$2.9x10^{-12}$
PeCDF, 1,2,3,7,8-	$4.7x10^{-14}$	$1.9x10^{-12}$	$2.6x10^{-14}$	$1.0x10^{-12}$
PeCDF, 2,3,4,7,8-	$8.8x10^{-13}$	$3.5x10^{-11}$	$4.7x10^{-13}$	$1.9x10^{-11}$
HxCDF, 1,2,3,4,7,8-	$1.3x10^{-12}$	$5.2x10^{-11}$	$6.5x10^{-13}$	$2.6x10^{-11}$
HxCDF, 1,2,3,6,7,8-	$2.1x10^{-13}$	$8.2x10^{-12}$	$1.0x10^{-13}$	$4.1x10^{-12}$
HxCDF, 1,2,3,7,8,9-	$2.4x10^{-14}$	$9.3x10^{-13}$	$1.2x10^{-14}$	$4.7x10^{-13}$
HxCDF, 2,3,4,6,7,8-	$7.2x10^{-14}$	$2.9x10^{-12}$	$3.6x10^{-14}$	$1.4x10^{-12}$
HpCDF, 1,2,3,4,6,7,8-	$2.9x10^{-13}$	$1.1x10^{-11}$	$1.2x10^{-13}$	$4.9x10^{-12}$
HpCDF, 1,2,3,4,7,8,9-	$6.9x10^{-15}$	$2.7x10^{-13}$	$3.0x10^{-15}$	$1.2x10^{-13}$
OCDF	$3.1x10^{-14}$	$1.2x10^{-12}$	$8.5x10^{-15}$	$3.4x10^{-13}$
PAHs				
Benzo(a)anthracene	$2.4x10^{-11}$	$9.3x10^{-10}$	$1.4x10^{-12}$	$5.6x10^{-11}$
Benzo(a)pyrene	$2.4x10^{-10}$	$9.4x10^{-9}$	$1.3x10^{-11}$	$5.4x10^{-10}$
Benzo(b)fluoranthene	$2.4x10^{-11}$	$9.1x10^{-10}$	$1.3x10^{-12}$	$5.4x10^{-11}$
Benzo(k)fluoranthene	$2.4x10^{-12}$	$9.4x10^{-11}$	$1.3x10^{-13}$	$5.4x10^{-12}$
Chrysene	$2.7x10^{-13}$	$1.1x10^{-11}$	$1.6x10^{-14}$	$6.6x10^{-13}$
Dibenzo(a,h)anthracene	$5.7x10^{-11}$	$2.2x10^{-9}$	$1.3x10^{-12}$	$5.4x10^{-11}$
Indeno(1,2,3-c,d)pyrene	$1.3x10^{-11}$	$5.0x10^{-10}$	$4.2x10^{-13}$	$1.7x10^{-11}$
PCB Coplanars				
TetraCB, 3,3',4,4'- (IUPAC #77)	$2.7x10^{-12}$	$1.1x10^{-10}$	$1.5x10^{-12}$	$5.9x10^{-11}$
PentaCB, 2',3,4,4',5- (IUPAC #123)	$1.8x10^{-13}$	$7.1x10^{-12}$	$9.8x10^{-14}$	$3.9x10^{-12}$
PentaCB, 2,3',4,4',5- (IUPAC #118)	$1.9x10^{-12}$	$7.3x10^{-11}$	$9.8x10^{-13}$	$3.9x10^{-11}$
PentaCB, 2,3,3',4,4'- (IUPAC #105)	$9.1x10^{-13}$	$3.6x10^{-11}$	$5.0x10^{-13}$	$2.0x10^{-11}$
PentaCB, 2,3,4,4',5- (IUPAC #114)	$5.7x10^{-14}$	$2.2x10^{-12}$	$3.1x10^{-14}$	$1.2x10^{-12}$
PentaCB, 3,3',4,4',5- (IUPAC #126)	$1.1x10^{-12}$	$4.3x10^{-11}$	$5.9x10^{-13}$	$2.4x10^{-11}$
HexaCB, 2,3',4,4',5,5'- (IUPAC #167)	$2.0x10^{-13}$	$7.8x10^{-12}$	$1.3x10^{-13}$	$4.1x10^{-12}$
HexaCB, 2,3,3',4,4',5'- (IUPAC #157)	$4.1x10^{-14}$	$4.6x10^{-12}$	$2.2x10^{-14}$	$8.8x10^{-13}$
HexaCB, 2,3,3',4,4',5- (IUPAC #156)	$1.4x10^{-13}$	$5.5x10^{-12}$	$7.2x10^{-14}$	$2.9x10^{-12}$
HexaCB, 3,3',4,4',5,5'- (IUPAC #169)	$2.4x10^{-14}$	$9.3x10^{-13}$	$1.1x10^{-14}$	$4.5x10^{-13}$
HeptaCB, 2,3,3',4,4',5,5'- (IUPAC #189)	$3.8x10^{-14}$	$1.5x10^{-12}$	$1.7x10^{-14}$	$6.7x10^{-13}$
Pesticides				
Aldrin	$5.4x10^{-10}$	$2.1x10^{-8}$	$2.9x10^{-10}$	$1.2x10^{-8}$
alpha-Chlordane	$1.0x10^{-9}$	$4.0x10^{-8}$	$5.6x10^{-10}$	$2.2x10^{-8}$
beta-BHC	$1.6x10^{-10}$	$6.3x10^{-9}$	$7.2x10^{-11}$	$2.9x10^{-9}$
DDD, 4,4'-	$7.4x10^{-9}$	$2.9x10^{-7}$	$3.9x10^{-9}$	$1.6x10^{-7}$
DDE, 4,4'-	$1.9x10^{-9}$	$7.4x10^{-8}$	$1.0x10^{-9}$	$4.1x10^{-8}$
DDT, 4,4'-	$2.2x10^{-9}$	$8.6x10^{-8}$	$1.2x10^{-9}$	$4.7x10^{-8}$
Dieldrin	$3.9x10^{-10}$	$1.5x10^{-8}$	$1.4x10^{-10}$	$5.8x10^{-9}$
gamma-Chlordane	$1.2x10^{-9}$	$4.7x10^{-8}$	$6.5x10^{-10}$	$2.6x10^{-8}$
Inorganics				
Arsenic	$9.2x10^{-7}$	$3.6x10^{-5}$	$1.6x10^{-6}$	$6.6x10^{-5}$
Beryllium	$7.5x10^{-8}$	$2.9x10^{-6}$	$1.3x10^{-7}$	$5.3x10^{-6}$
Semivolatiles				
Bis(2-ethylhexyl)phthalate	$2.2x10^{-9}$	$8.7x10^{-8}$	$1.2x10^{-10}$	$4.7x10^{-9}$

a. Exposure point used to calculate Lifetime Average Daily Intake for PCDD/Fs and coplanar PCBs were assessed as 2,3,7,8-TCDD equivalent concentrations.

b. RME = Reasonable Maximum Exposure

Table 3-6. Average Daily Intakes (Noncarcinogenic) Estimates (mg/kg-day)

Chemicals of Potential Concern	Blue Crab		Striped Bass	
	Typical	RME (a)	Typical	RME (a)
PAHs				
Acenaphthene	8.3×10^{-10}	9.9×10^{-9}	2.3×10^{-11}	2.8×10^{-10}
Acenaphthylene	6.1×10^{-10}	7.2×10^{-9}	1.8×10^{-11}	2.1×10^{-10}
Anthracene	1.0×10^{-9}	1.2×10^{-8}	3.6×10^{-11}	4.3×10^{-10}
Benzo(ghi)perylene	9.1×10^{-10}	1.1×10^{-8}	3.0×10^{-11}	3.7×10^{-10}
Dibenzofuran	7.4×10^{-10}	8.7×10^{-9}	2.1×10^{-11}	2.5×10^{-10}
Fluoranthene	3.9×10^{-9}	4.6×10^{-8}	2.2×10^{-10}	2.6×10^{-9}
Fluorene	8.1×10^{-10}	9.6×10^{-9}	2.4×10^{-11}	2.9×10^{-10}
Phenanthrene	2.5×10^{-9}	2.9×10^{-8}	8.6×10^{-11}	1.0×10^{-9}
Pyrene	3.7×10^{-9}	4.3×10^{-8}	2.0×10^{-10}	2.4×10^{-9}
PCBs				
Aroclor 1248	3.1×10^{-7}	3.7×10^{-6}	1.7×10^{-7}	2.0×10^{-6}
Aroclor 1254	7.6×10^{-8}	9.0×10^{-7}	4.1×10^{-8}	4.9×10^{-7}
Pesticides				
Aldrin	4.2×10^{-9}	5.0×10^{-8}	2.3×10^{-9}	2.7×10^{-8}
alpha-Chlordane	8.0×10^{-9}	9.4×10^{-8}	4.3×10^{-9}	5.2×10^{-8}
DDT, 4,4'-	1.7×10^{-8}	2.0×10^{-7}	9.1×10^{-9}	1.1×10^{-7}
delta-BHC	1.4×10^{-9}	1.7×10^{-8}	6.9×10^{-10}	8.3×10^{-9}
Dieldrin	3.0×10^{-9}	3.6×10^{-8}	1.1×10^{-9}	1.3×10^{-8}
Endrin	1.1×10^{-8}	1.2×10^{-7}	5.6×10^{-9}	6.7×10^{-8}
gamma-Chlordane	9.3×10^{-9}	1.1×10^{-7}	5.1×10^{-9}	6.1×10^{-8}
Methoxychlor	1.9×10^{-8}	2.3×10^{-7}	1.0×10^{-8}	1.3×10^{-7}
Inorganics				
Aluminum	0.0069	0.081	0.012	0.15
Antimony	4.7×10^{-6}	5.6×10^{-5}	8.4×10^{-6}	0.00010
Arsenic	7.2×10^{-6}	8.5×10^{-5}	1.3×10^{-5}	0.00015
Barium	0.00011	0.0013	0.00019	0.0023
Beryllium	5.8×10^{-7}	6.9×10^{-6}	1.0×10^{-6}	1.2×10^{-5}
Cadmium	3.4×10^{-6}	4.0×10^{-5}	6.0×10^{-6}	7.3×10^{-5}
Chromium	3.4×10^{-6}	4.0×10^{-5}	6.0×10^{-6}	7.3×10^{-5}
Cobalt	7.1×10^{-6}	8.4×10^{-5}	1.3×10^{-5}	0.00015
Copper	0.00012	0.0014	0.00022	0.0026
Manganese	0.00020	0.0024	0.00036	0.0044
Mercury	1.8×10^{-6}	2.2×10^{-5}	3.3×10^{-6}	3.9×10^{-5}
Nickel	3.1×10^{-5}	0.00036	5.5×10^{-5}	0.00066
Selenium	7.5×10^{-7}	8.9×10^{-6}	1.3×10^{-6}	1.6×10^{-5}
Silver	3.3×10^{-6}	4.0×10^{-5}	5.9×10^{-6}	7.2×10^{-5}
Thallium	3.0×10^{-7}	3.5×10^{-6}	5.3×10^{-7}	6.4×10^{-6}
Vanadium	2.0×10^{-5}	0.00024	6.3×10^{-5}	0.00043
Zinc	0.00030	0.0035	0.00052	0.0063
Semivolatiles				
Bis(2-ethylhexyl)phthalate	1.7×10^{-8}	2.0×10^{-7}	9.1×10^{-10}	1.1×10^{-8}
Butyl benzyl phthalate	6.4×10^{-10}	7.5×10^{-9}	3.8×10^{-11}	4.6×10^{-10}
Di-n-butyl phthalate	6.9×10^{-10}	8.1×10^{-9}	4.1×10^{-11}	4.9×10^{-10}
Di-n-octyl phthalate	8.6×10^{-10}	1.0×10^{-8}	5.3×10^{-11}	6.4×10^{-10}
Trichlorobenzene, 1,2,4-	7.1×10^{-10}	8.4×10^{-9}	2.2×10^{-11}	2.6×10^{-10}
Miscellaneous				
Dibutyltin	1.6×10^{-7}	1.9×10^{-6}	2.8×10^{-7}	3.4×10^{-6}
Monobutyltin	2.2×10^{-7}	2.6×10^{-6}	7.9×10^{-7}	9.5×10^{-6}

a. RME = Reasonable Maximum Exposure

3.4 Toxicity Assessment

Toxicity assessment is defined by the EPA (1989a) as an evaluation of the inherent toxicologic potential associated with exposure to a chemical. Toxicity assessment is a two–step process that includes hazard identification and dose–response assessment. Whereas hazard identification is a qualitative description of the potential health effects associated with exposure to a given chemical, dose–response assessment is a quantitative analysis of the relationship between the magnitude of the dose received and the observed toxicologic responses in an exposed population (EPA, 1989a). In an ideal situation, actual human data would be used to quantitatively characterize the potential occurrence of adverse effects. In most instances, however, such data are not available. Therefore, the scientific understanding of the dose–response relationship is largely based on data collected from animal studies (usually rodent bioassays) and hypotheses about what might occur in humans. Mathematical models are used to estimate the possible responses in humans at levels far below those tested in animals. These models contain several limitations which should be considered when risk estimates are evaluated (EPA, 1989a) as discussed in Section 3.5.3.

In an effort to determine whether exposure to a chemical can cause an increase in the incidence of a particular adverse health effect and whether the adverse health effect is likely to occur in humans, the nature and strength of causation are characterized by the EPA according to the "weight–of–evidence" carcinogen classification system (EPA, 1989a). This classification system is summarized in Table 3-7. The EPA weight–of–evidence carcinogen classification for each CPC is summarized in Table 3-8.

Information for each CPC used to evaluate chemical hazards was obtained from one of the following sources: the EPA IRIS, the Health Effects Assessment Summary Tables (HEAST) (EPA, 1994a), EPA criteria documents, or EPA's ECAO[now NCEA]. IRIS contains descriptive and quantitative toxicity information and is considered to be the most authoritative source of verified EPA dose–response values, including CSFs and RfDs for supporting risk assessments (EPA, 1989a). Although IRIS values are recommended by the agency to ensure consistency in risk assessments, it is important to note that alternative toxicity values may also be used in Superfund risk assessments if they are based upon more recent, credible, or relevant toxicological data (EPA, 1993b). For the purpose of this conservative screening–level HHRA, however, EPA–derived toxicity values were used for all chemicals.

Table 3-7. EPA Weight-of-Evidence Classification System for Carcinogenicity

Group	Description
A	Human carcinogen
B1 or B2	Probable human carcinogen B1 indicates that limited human data are available B2 indicates sufficient evidence in animals and inadequate or no evidence in humans
C	Possible human carcinogen
D	Not classifiable as to human carcinogenicity
E	Evidence of noncarcinogenicity for humans

Source: EPA 1989a.

Table 3-8. Oral Toxicity Values for Potential Carcinogenic Effects

Chemical	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$	EPA Weight of Evidence Classification	Type of Tumor	Method of Administration	Source
Semivolatiles					
Bis(2-ethylhexyl)phthalate	0.014	B2	hepatocellular carcinomas and adenomas	diet	EPA, 1995
Butyl benzyl phthalate	NA	C			EPA, 1995
Dimethylphthalate	NA	D			
Di-n-butyl phthalate	NA	D			EPA, 1995
Di-n-octyl phthalate	NA				
Trichlorobenzene, 1,2,4-	NA	D			EPA, 1995
Inorganics					
Aluminum	NA	D			ECAO, 1995
Antimony	NA				
Arsenic	1.75	A	skin	drinking water	EPA, 1995
Barium	NA				
Beryllium	4.3	B2	gross tumors	drinking water	EPA, 1995
Cadmium	NA	B1			EPA, 1995
Chromium	NA	A			EPA, 1995 Based on Chromium VI*
Cobalt	NA				
Copper	NA	D			EPA, 1995
Cyanide	NA	D			EPA, 1995
Lead	NA	B2			EPA, 1995
Manganese	NA	D			EPA, 1995
Mercury, methyl	NA				
Nickel	NA				
Selenium	NA	D			EPA, 1995
Silver	NA	D			EPA, 1995
Thallium	NA				
Titanium	NA				
Vanadium	NA				
Zinc	NA	D			EPA, 1995

Table 3-8. Oral Toxicity Values for Potential Carcinogenic Effects

Chemical	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$	EPA Weight of Evidence Classification	Type of Tumor	Method of Administration	Source
PAHs					
Acenaphthene	NA				
Acenaphthylene	NA	D			EPA, 1995
Anthracene	NA	D			EPA, 1995
Benzo(a)anthracene	7.3	B2	forestomach papillomas and carcinomas	diet	Based on Benzo(a)pyrene*
Benzo(a)pyrene	7.3	B2	forestomach papillomas and carcinomas	diet	EPA, 1995
Benzo(b)fluoranthene	7.3	B2	forestomach papillomas and carcinomas	diet	Based on Benzo(a)pyrene*
Benzo(ghi)perylene	NA	D			EPA, 1995
Benzo(k)fluoranthene	7.3	B2	forestomach papillomas and carcinomas	diet	Based on Benzo(a)pyrene*
Carbazole	0.02	B2	liver	diet	EPA, 1994a
Chrysene	7.3	B2	forestomach papillomas and carcinomas	diet	Based on Benzo(a)pyrene*
Dibenzo(a,h)anthracene	7.3	B2	forestomach papillomas and carcinomas	diet	Based on Benzo(a)pyrene*
Dibenzofuran	NA	D			EPA, 1995
Fluoranthene	NA	D			EPA, 1995
Fluorene	NA	D			EPA, 1995
Indeno(1,2,3-c,d)pyrene	7.3	B2	forestomach papillomas and carcinomas	diet	Based on Benzo(a)pyrene*
Methylnaphthalene,2-	NA	D			ECAO, 1995
Phenanthrene	NA	D			EPA, 1995
Pyrene	NA	D			EPA, 1995
Pesticides					
Aldrin	17	B2	liver carcinoma	diet	EPA, 1995
beta-BHC	1.8	C	benign liver tumors	diet	EPA, 1995
Chlordane	1.3	B2	hepatocellular carcinoma	diet	EPA, 1995
alpha-Chlordane	1.3	B2	hepatocellular carcinoma	diet	Based on Chlordane*
gamma-Chlordane	1.3	B2	hepatocellular carcinoma	diet	Based on Chlordane*
DDD, 4,4'-	0.24	B2	liver	diet	EPA, 1995
DDE, 4,4'-	0.34	B2	liver/thyroid	diet	EPA, 1995
DDT, 4,4'-	0.34	B2	liver	diet	EPA, 1995
delta-BHC	NA	D			EPA, 1995
Dieldrin	16	B2	liver carcinoma	diet	EPA, 1995
Endosulfan I	NA				
Endosulfan II	NA				
Endosulfan sulfate	NA				
Endrin	NA	D			EPA, 1995
Endrin aldehyde	NA				
Endrin ketone	NA				
Heptachlor epoxide (exo)	9.10	B2	hepatocellular carcinomas	diet	EPA, 1995
Methoxychlor	NA	D			

Table 3-8. Oral Toxicity Values for Potential Carcinogenic Effects

Chemical	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$	EPA Weight of Evidence Classification	Type of Tumor	Method of Administration	Source
Polychlorinated Biphenyls					
TetraCB, 3,3',4,4'- (IUPAC #77)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
PentaCB, 2',3,4,4',5- (IUPAC #123)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
PentaCB, 2,3',4,4',5- (IUPAC #118)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
PentaCB, 2,3,3',4,4'- (IUPAC #105)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
PentaCB, 2,3,4,4',5- (IUPAC #114)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
PentaCB, 3,3',4,4',5- (IUPAC #126)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HexaCB, 2,3',4,4',5,5'- (IUPAC #167)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HexaCB, 2,3,3',4,4',5'- (IUPAC #157)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HexaCB, 2,3,3',4,4',5- (IUPAC #156)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HexaCB, 3,3',4,4',5,5'- (IUPAC #169)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HeptaCB, 2,3,3',4,4',5,5'- (IUPAC #189)	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
Aroclor 1248	7.7	B2	trabecular carcinoma/ adenocarcinoma	diet	EPA, 1995
Aroclor 1254	7.7	B2	trabecular carcinoma/ adenocarcinoma	diet	EPA, 1995
Polychlorinated Dibenzo-p-dioxins and Dibenzofurans					
TCDD, 2,3,7,8-	75,000	B2	liver and respiratory system tumors	diet	EPA, 1991
PeCDD, 1,2,3,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HxCDD, 1,2,3,4,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HxCDD, 1,2,3,6,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HxCDD, 1,2,3,7,8,9-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HpCDD, 1,2,3,4,6,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
OCDD	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
TCDF, 2,3,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
PeCDF, 1,2,3,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
PeCDF, 2,3,4,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HxCDF, 1,2,3,4,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HxCDF, 1,2,3,6,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HxCDF, 1,2,3,7,8,9-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HxCDF, 2,3,4,6,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HpCDF, 1,2,3,4,6,7,8-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
HpCDF, 1,2,3,4,7,8,9-	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
OCDF	75,000		liver and respiratory system tumors	diet	Based on 2,3,7,8-TCDD*
Miscellaneous					
TEPH	NA				
Dichloroethene, 1,2- (total)	NA				
Dibutyltin	NA	D			ECAO, 1995
Monobutyltin	NA	D			ECAO, 1995

*Surrogate toxicity value for chemical with no IRIS or HEAST toxicity value. NA- Not available.

HEAST is prepared annually by ECAO and provides information on chemicals commonly found at both Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and Resource Conservation and Recovery Act (RCRA) sites. In addition to verified toxicity values, HEAST lists interim CSFs and RfDs. For this assessment, information contained in IRIS superseded all other sources of information and other sources were consulted only when information was not available in IRIS. Consistent with EPA (1989a) guidance, EPA criteria documents (i.e., EPA, 1991b) were also consulted as sources of toxicity information for chemicals without published values in IRIS.

Consistent with EPA (1989a) guidance, ECAO was consulted directly to obtain provisional toxicity information for chemicals for which no toxicity values were reported in IRIS or HEAST or other EPA documents. Toxicity information for aluminum, cobalt, organotins, and dibenzofuran was obtained from ECAO. Individual sources of information on particular CPC are provided in Tables 3-8 and 3-9, as well as in individual chemical toxicity profiles that are provided in Appendix D. For a limited number of chemicals (1,2–dichloroethene, dimethylphthalate, lead, and titanium), toxicity values were not available from any of the sources discussed above. Consistent with EPA (1989a), these chemicals were retained as CPC, and were considered in the assessment of uncertainties in the risk assessment.

The results of the toxicity assessment for the CPC identified in Section 3.1 are discussed in toxicity profiles provided in Appendix D. In cases where a group of chemicals share similar physical, chemical, and/or toxicologic properties, such as PAHs and PCBs, a single toxicity profile was prepared for all the chemicals belonging to that group. Toxicity information for CPC is also summarized in Tables 3-8 and 3-9. For the purposes of this screening-level HHRA, the oral slope factor for inorganic arsenic has been used to characterize carcinogenic risk associated with consumption of arsenic in fish and crab tissue. It is suspected that a portion of the arsenic in fish is typically comprised of arsenobetaine, a quaternary methylated derivative of arsenic. Unlike inorganic arsenic, mono- and di-methylated forms of arsenic have been found to be negative in cancer bioassay systems and are not classified by EPA as carcinogenic. However, arsenobetaine has never been tested for carcinogenicity. In addition, there are reports of inorganic arsenic

Table 3-9. Oral Toxicity Values for Potential Noncarcinogenic Effects

Chemical	Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Method of Administration	Combined Uncertainty and Modifying Factors	Source
Semivolatiles						
Bis(2-ethylhexyl)phthalate	0.02	medium	increased relative liver weight	diet	1000	EPA,1995
Butyl benzyl phthalate	0.2	low	increased liver to body weight and liver to brain weight ratios	diet	1000	EPA, 1995
Dimethylphthalate	NA					
Di-n-butyl phthalate	0.1	low	mortality	diet	1000	EPA, 1995
Di-n-octyl phthalate	0.02		increased liver and kidney weights, increased SGOT activity	diet	1000	EPA, 1994A
Trichlorobenzene, 1,2,4-	0.01	medium	increased adrenal weight	drinking water	1000	EPA, 1995
Inorganics						
Aluminum	1	low	neurotoxicity	gavage	100	ECAO, 1995
Antimony	0.0004	low	longevity, blood glucose, cholesterol	drinking water	1000	EPA, 1995
Arsenic	0.0003	medium	hyperpigmentation, keratosis, vascular complications	drinking water	3	EPA, 1995
Barium	0.07	medium	increased blood pressure	drinking water	3	EPA, 1995
Beryllium	0.005	low	none	drinking water	100	EPA, 1995
Cadmium	0.001	high	proteinuria	diet	10	EPA, 1995
Chromium	0.005	low	none	drinking water	500	EPA, 1995 Based on Chromium VI*
Cobalt	0.06	not applicable	polycythemia	diet	NA	ECAO, 1995
Copper	0.037		gastrointestinal irritation	water	NA	EPA, 1994A
Cyanide	0.02	medium	weight loss, thyroid effects, myelin degeneration	diet	500	EPA, 1995
Lead	NA					
Manganese	0.14	not applicable	CNS effects	diet	1	EPA, 1995
Mercury	0.0003		kidney effects	parenteral	1000	EPA, 1994A
Nickel	0.02	medium	decreased body and organ weights	diet	300	EPA, 1995
Selenium	0.005	high	selenosis	diet	3	EPA, 1995
Silver	0.005	low	argyria	intravenous	3	EPA, 1995
Thallium	0.00007	low	increased SGOT and LDH	gavage	3000	EPA, 1995 Based on Thallium chloride*
Titanium	NA					
Vanadium	0.007		not specified	drinking water	100	EPA, 1994A
Zinc	0.3	medium	ESOD decrease	diet	3	EPA, 1995

Table 3-9. Oral Toxicity Values for Potential Noncarcinogenic Effects

Chemical	Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Method of Administration	Combined Uncertainty and Modifying Factors	Source
PAHs						
Acenaphthene	0.06	low	hepatotoxicity	gavage	3000	EPA, 1995
Acenaphthylene	0.03	low	kidney effects	gavage	3000	Based on Pyrene*
Anthracene	0.3	low	none	gavage	3000	EPA, 1995
Benzo(a)anthracene	NA					
Benzo(a)pyrene	NA					
Benzo(b)fluoranthene	NA					
Benzo(ghi)perylene	0.03	low	kidney effects	gavage	3000	Based on Pyrene*
Benzo(k)fluoranthene	NA					
Carbazole	NA					
Chrysene	NA					
Dibenzo(a,h)anthracene	NA					
Dibenzofuran	0.004	low	kidney effects	diet	3000	ECAO, 1995
Fluoranthene	0.04	low	nephropathy, increased liver weight, hematological alterations	gavage	3000	EPA, 1995
Fluorene	0.04	low	decreased red blood cells	gavage	3000	EPA, 1995
Indeno(1,2,3-c,d)pyrene	NA					
Methylnaphthalene, 2-	NA					ECAO, 1995
Phenanthrene	0.03	low	kidney effects	gavage	3000	Based on Pyrene*
Pyrene	0.03	low	kidney effects	gavage	3000	EPA, 1995
Pesticides						
Aldrin	0.00003	medium	liver toxicity	diet	1000	EPA, 1995
beta-BHC	NA					
Chlordane	0.00006	low	regional liver hypertrophy	diet	1000	EPA, 1995
alpha-Chlordane	0.00006	low	regional liver hypertrophy	diet	1000	Based on Chlordane*
gamma-Chlordane	0.00006	low	regional liver hypertrophy	diet	1000	Based on Chlordane*
DDD, 4,4'-	NA					
DDE, 4,4'-	NA					
DDT, 4,4'-	0.0005	medium	liver lesions	diet	100	EPA, 1995
delta-BHC	0.0003	medium	liver and kidney toxicity	diet	1000	Based on Gamma-HCH*
Dieldrin	0.00005	medium	liver lesions	diet	100	EPA, 1995
Endosulfan I	0.006	medium	reduced body weight gain, glomerulonephrosis, blood vessel aneurysms	diet	100	Based on Endosulfan*
Endosulfan II	0.006	medium	reduced body weight gain, glomerulonephrosis, blood vessel aneurysms	diet	100	Based on Endosulfan*
Endosulfan sulfate	0.006	medium	reduced body weight gain, glomerulonephrosis, blood vessel aneurysms	diet	100	Based on Endosulfan*
Endrin	0.0003	medium	liver lesions; convulsions	diet	100	EPA, 1995
Endrin aldehyde	0.0003	medium	liver lesions; convulsions	diet	100	Based on Endrin*
Heptachlor epoxide (exo)	0.000013	low	increased liver to body weight ratio	diet	1000	EPA, 1995 EPA, 1995 Based on Heptachlor epoxide*
Methoxychlor	0.005	low	loss of litters	not specified	1000	EPA, 1995

Table 3-9. Oral Toxicity Values for Potential Noncarcinogenic Effects

Chemical	Chronic Oral RfD (mg/kg-day)	Confidence Level	Critical Effect	Method of Administration	Combined Uncertainty and Modifying Factors	Source
Polychlorinated Biphenyls						
TetraCB, 3,3',4,4'- (IUPAC #77)	NA					
PentaCB, 2',3,4,4',5- (IUPAC #123)	NA					
PentaCB, 2,3',4,4',5- (IUPAC #118)	NA					
PentaCB, 2,3,3',4,4'- (IUPAC #105)	NA					
PentaCB, 2,3,4,4',5- (IUPAC #114)	NA					
PentaCB, 3,3',4,4',5- (IUPAC #126)	NA					
HexaCB, 2,3',4,4',5,5'- (IUPAC #167)	NA					
HexaCB, 2,3,3',4,4',5'- (IUPAC #157)	NA					
HexaCB, 2,3,3',4,4',5- (IUPAC #156)	NA					
HexaCB, 3,3',4,4',5,5'- (IUPAC #169)	NA					
HeptaCB, 2,3,3',4,4',5,5'- (IUPAC #189)	NA					
Aroclor 1248	0.00002	medium	ocular exudate, meibomian gland inflammation	capsule	300	Based on Aroclor 1254*
Aroclor 1254	0.00002	medium	ocular exudate, meibomian gland inflammation	capsule	300	EPA, 1995
Polychlorinated Dibenzo-p-dioxins and Dibenzofurans						
TCDD, 2,3,7,8-	NA					
PeCDD, 1,2,3,7,8-	NA					
HxCDD, 1,2,3,4,7,8-	NA					
HxCDD, 1,2,3,6,7,8-	NA					
HxCDD, 1,2,3,7,8,9-	NA					
HpCDD, 1,2,3,4,6,7,8-	NA					
OCDD	NA					
TCDF, 2,3,7,8-	NA					
PeCDF, 1,2,3,7,8-	NA					
PeCDF, 2,3,4,7,8-	NA					
HxCDF, 1,2,3,4,7,8-	NA					
HxCDF, 1,2,3,6,7,8-	NA					
HxCDF, 1,2,3,7,8,9-	NA					
HxCDF, 2,3,4,6,7,8-	NA					
HpCDF, 1,2,3,4,6,7,8-	NA					
HpCDF, 1,2,3,4,7,8,9-	NA					
OCDF	NA					
Miscellaneous						
TEPH	NA					
Dichloroethene, 1,2-(total)	NA					
Dibutyltin	0.00003	low	depressed immunity	diet	not given	ECAO, 1995
Monobutyltin	0.00003	low	depressed immunity	diet	not given	ECAO, 1995

*Surrogate toxicity value for chemical with no IRIS or HEAST toxicity value. NA-Not available.

comprising over 40% of the total arsenic in a fish tissue sample (Lunde, 1977). Therefore, in the absence of evidence to the contrary, it seems prudent to apply the inorganic arsenic oral slope factor to derive a carcinogenic risk for consumed arsenic in this HHRA.

3.4.1 Noncarcinogenic Response

It is widely accepted in the scientific community that most biological effects related to exposure to chemical substances occur only after a threshold dose has been achieved (Klaassen et al., 1986). A threshold dose is a level of intake below which adverse effects are not expected to occur. For the purposes of establishing toxicological benchmarks for noncarcinogenic chemicals, the threshold dose is usually estimated from the no–observed–adverse–effect–level (NOAEL) or the lowest–observed– adverse–effect–level (LOAEL) determined in chronic animal studies. The NOAEL is defined as the highest dose at which no adverse effects occur, while the LOAEL is defined as the lowest dose at which adverse effects are observed.

NOAELs and LOAELs derived from both animal and human studies are used by EPA to establish chronic reference doses (RfDs) for humans. EPA (1989a) defines a chronic RfD as "an estimate (with uncertainty spanning an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime." Uncertainty factors are incorporated into RfDs in an attempt to account for limitations in the quality or quantity of available data. Many RfDs include a 100–fold safety factor to account for uncertainties in extrapolating animal data to human health effects (a factor of ten) and differences in sensitivity within the human population (another factor of ten). However, if the available databases are incomplete, an additional uncertainty factor, known as a modifying factor, may be applied. For example, if available data do not establish a NOAEL and/or there are data gaps for some types of health effects, a safety factor of 1,000, representing an uncertainty factor of 100 and a modifying factor of 10, could be used to establish the RfD.

Table 3-9 presents the health criteria used to evaluate noncarcinogenic effects resulting from oral exposures. For the purposes of this screening–level HHRA, the oral reference doses established by EPA were used as the basis for assessing the potential noncarcinogenic chronic health hazards for the hypothetically exposed populations.

3.4.2 Carcinogenic Response

Both human epidemiological studies and animal bioassays are used to assess the carcinogenic potential of many chemicals. Frequently, epidemiological studies have been conducted for occupational populations because they are typically exposed to higher concentrations of chemicals than the general population. Animal carcinogenicity bioassays involve measuring the tumor incidence in rats or mice following administration of various doses of the chemical for the lifetime of the animal.

For regulatory purposes, it is assumed that any dose of a carcinogen, no matter how small, presents some level of risk. To estimate the theoretically potential response at these low doses, various mathematical models have been developed. The accuracy of the projected risk at the dose of interest is a function of the accuracy of the mathematical model in predicting the true (but not measurable) relationship between dose and risk at low dose levels. EPA generally uses the linearized multistage (LMS) model for low dose extrapolation from animal studies (Munro and Krewski, 1981). This model assumes that the slope of a dose–response curve can be extrapolated to zero in a linear manner.

The numerical expression for the carcinogenic potency of a chemical calculated by the LMS model is known as the q_1^*, or cancer slope factor. The q_1^* represents the 95 percent upper confidence limit on the slope of a dose–response curve derived from either animal or human studies. The slope of the dose–response curve is a quantitative estimate of a chemical's carcinogenic potency and is calculated as the change in tumor incidence (y-axis) over the change in dose (x–axis). Thus, the units of q_1^* are the probability of tumor incidence divided by the dose level given in milligrams (mg) of chemical per kilogram (kg) of body weight per day ($[mg/kg–day]^{-1}$).

Cancer slope factors are considered to be theoretical upper bound estimates of risk at a 95 percent upper confidence level (i.e., there is a 95 percent probability that the true risks do not exceed these levels and are likely to be much lower). The Human Health Assessment Group (HHAG), formerly called Carcinogen Assessment Group, states that the use of the LMS model and upper–bound risk estimates is appropriate, but the lower limit of risk may be as low as zero (EPA, 1986). When physiological factors (i.e., mechanisms of action, metabolism) are considered, the

best estimate of the risk at very low levels may indeed be zero. The HHAG stated that an "established procedure does not yet exist for making 'most likely' or 'best' estimates of risk within the range of uncertainty defined by the upper and lower limit estimates" (EPA, 1986).

Regulatory agencies in the United States continue to base CSFs on the nonthreshold LMS model (EPA, 1989a). For the purposes of this screening–level assessment, this procedure was followed, and CSFs established by EPA (presented in Table 3-8) were used to assess the potential carcinogenic risks to hypothetically exposed populations.

For certain classes of compounds that are structurally similar, and assumed to be carcinogenic, and for which data are insufficient to calculate individual CSFs, regulatory agencies have adopted, as an interim procedure, the use of TEF schemes (EPA, 1989d, 1993c, 1994b). The rationale for using such a scheme is to predict the carcinogenic potency for those compounds for which chronic carcinogenicity bioassays have not been conducted. The TEF methodology estimates the toxicity of each compound within a defined chemical class relative to a reference compound for which adequate dose response data are available. Application of TEFs, in theory, allows the concentration of each individual compound or congener to be converted into an equivalent concentration of the reference compound for use in assessing risks. In general, TEFs are estimated from the results of short–term *in vivo* and *in vitro* toxicity bioassays (EPA, 1989d, 1993c, 1994b).

TEF schemes have been used by the EPA for evaluation of compounds such as PCDD/Fs, carcinogenic PAHs, and coplanar PCBs (EPA, 1989d, 1993c, 1994a). Consistent with EPA practice, TEFs have been used in this assessment to evaluate the carcinogenic potential of PCDD/Fs, carcinogenic PAHs, and coplanar PCBs. For PCDD/Fs, EPA currently employs the International Toxic Equivalency Factor or I–TEF scheme, and assigns a TEF to each congener based on its assumed toxicity relative to that of 2,3,7,8–TCDD (Table 3-10) (EPA, 1989d). However, as described in further detail in the uncertainty analysis (Section 3.5.3) recent evidence demonstrates that the biological activity of PCDD/F congeners in environmental media may be far less than additive. Specifically, the expected biological activity of the PCDD/F mixture (when the TEF approach is applied as above) may be much greater than measured in an *in vitro* or *in vivo* assay system. Hence, because additivity was assumed in this assessment, the PCDD/F-related risks described in this HHRA are likely over-estimated. For carcinogenic PAHs, interim oral potencies are based on the estimated carcinogenic potency relative to benzo(a)pyrene (BaP) (Table

Table 3-10. International Toxicity Equivalency Factors (TEFs) for PCDDs and PCDFs

Compound	TEF
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	0.5
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HXCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.001
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.001

Source: EPA, 1989d.

3-11) (EPA, 1993c). For coplanar PCBs, a TEF scheme developed by Safe (1990) (Table 3-12), and used by EPA (1994b) in its recent Draft Health Assessment Document (HAD) for dioxin and related compounds, was used to evaluate coplanar PCBs.

3.5 Human Health Risk Characterization

Risk characterization integrates all aspects of the health risk assessment process and provides a scientific interpretation of the overall assessment (EPA, 1989a, 1992). In the risk characterization, all data, results, conclusions, and uncertainties from the hazard identification, dose-response assessment, and exposure assessment are evaluated in order to draw scientifically supportable conclusions. Not only are the potential noncarcinogenic and carcinogenic health risks quantified (NAS, 1983), but also, the risk characterization presents and discusses the critical uncertainties of the analysis (EPA, 1992). Recent EPA (1992) guidelines specify that four critical tasks be accomplished by the risk characterization. According to EPA, "the risk characterization:

1. Integrates the individual characterizations from the hazard identification, dose-response, and exposure assessments;

2. Provides an evaluation of the overall quality of the assessment and the degree of confidence the authors have in the estimates of risk and conclusions drawn;

3. Describes risks to individuals and population in terms of extent and severity of probable harm; and

4. Communicates results of the risk assessment to the risk manager" (EPA, 1992).

In this risk characterization, chemical-specific, hypothetical risk estimates are presented. Consistent with EPA guidance (1989a), the potential exposures have been combined across exposure pathways and across the various CPC in order to present upper-bound, cumulative carcinogenic and noncarcinogenic risk estimates. As stated in the IWP, cumulative risks and hazard indices are compared to acceptable benchmark levels supported by EPA (1990a). In keeping with the most recent EPA (1992) guidance, professional judgement has been relied upon to select the most significant uncertainties (those that define and explain the risk estimates) for discussion in this risk characterization.

Table 3-11. Interim Relative Potency Factors for PAHs

Compound	Relative Potency
Benzo(a)pyrene	1.0
Benzo(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.01
Chrysene	0.001
Dibenzo(a,h)anthracene	1.0
Indeno(1,2,3-cd)pyrene	0.1

Source: EPA, 1993c.

Table 3-12. Toxicity Equivalency Factors for Coplanar PCBs

Compound	TEF
1. Coplanar PCBs	
PentaCB, 3,3',4,4',5- (IUPAC #126)	0.1
HexaCB, 3,3',4,4',5,5'- (IUPAC #169)	0.05
TetraCB, 3,3',4,4'- (IUPAC #77)	0.01
2. Monortho coplanar PCBs	
PentaCB, 2,3',4,4',5- (IUPAC #118)	0.001
PentaCB, 2,3,3',4,4'- (IUPAC #105)	0.001
PentaCB, 2',3,4,4',5- (IUPAC #123)	0.001
PentaCB, 2,3,4,4',5- (IUPAC #114)	0.001
HexaCB, 2,3,3',4,4',5- (IUPAC #156)	0.001
HexaCB, 2,3,3',4,4',5'- (IUPAC #157)	0.001
HexaCB, 2,3',4,4',5,5'- (IUPAC #167)	0.001
HeptaCB, 2,3,3',4,4',5,5'- (IUPAC #189)	0.001
3. Diortho coplanar PCBs	0.00002

Safe, 1990.

3.5.1 Carcinogenic Risk

Upper-bound incremental lifetime cancer risks were estimated for human exposure to bioaccumulative chemicals from ingestion of striped bass and blue crab. Risks were calculated by multiplying estimated doses, expressed as LADIs calculated in Section 3.3.3, by the appropriate toxicity values, as reported in Section 3.4. The equation for estimating cancer risk is as follows:

$$Risk = LADI \times CSF$$

where:

Risk = Lifetime incremental cancer risk (unitless; expressed as a probability);
LADI = Lifetime average daily intake (mg/kg-day); and
CSF = Cancer slope factor $(mg/kg\text{-}day)^{-1}$.

This equation yields an approximation of incremental cancer risk above the background cancer rate of 33 in 100 (ACS, 1993).

Cumulative risks were then calculated by summing across all chemicals to determine the total incremental lifetime cancer risk for individuals consuming striped bass and blue crab. Consistent with EPA (1992) guidance, risk estimates for both the typical and the RME situations are presented. The chemical-specific carcinogenic risk estimates for typical and RME exposures resulting from the consumption of blue crab and striped bass are reported in Table 3-13. The total incremental lifetime cancer risk for combined consumption of both blue crab and striped bass is estimated to be 7×10^{-6} for typical exposure and 3×10^{-4} for the RME. In comparison, the range of incremental cancer risk considered acceptable for CERCLA sites is 1×10^{-6} to 1×10^{-4} (one in a million to one in ten thousand) (EPA, 1990b).

Chemicals contributing greater than 1% of the total risk, in order of contribution are: arsenic (62%); beryllium (12%); 2,3,7,8-TCDD (9.0%); 3,3',4,4'-TetraCB (4.3%); 2,3',4,4',5-PentaCB (2.9%); 1,2,3,4,7,8-HxCDF (2.0%); 3,3',4,4',5-PentaCB (1.7%); 2,3,3',4,4'-PentaCB (1.5%); and 2,3,4,7,8-PeCDF (1.4%). Collectively, these chemicals represent 97 percent of the total carcinogenic risks estimated in this assessment (Table 3-14).

Table 3-13. Hypothetical Cancer Risks Associated with Consumption of Blue Crab and Striped Bass

Chemicals of Potential Concern	Blue Crab Risks		Striped Bass Risks		Total Risks	
	Typical	RME (a)	Typical	RME (a)	Typical	RME (a)
Inorganics						
Arsenic	$2x10^{-6}$	$6x10^{-5}$	$3x10^{-6}$	$1x10^{-4}$	$4x10^{-6}$	$2x10^{-4}$
Beryllium	$3x10^{-7}$	$1x10^{-5}$	$6x10^{-7}$	$2x10^{-5}$	$9x10^{-7}$	$4x10^{-5}$
PCDD/Fs						
TCDD, 2,3,7,8-	$4x10^{-7}$	$2x10^{-5}$	$2x10^{-7}$	$9x10^{-6}$	$7x10^{-7}$	$3x10^{-5}$
PeCDD, 1,2,3,7,8-	$4x10^{-9}$	$2x10^{-7}$	$2x10^{-9}$	$8x10^{-8}$	$6x10^{-9}$	$2x10^{-7}$
HxCDD, 1,2,3,4,7,8-	$8x10^{-10}$	$3x10^{-8}$	$4x10^{-10}$	$1x10^{-8}$	$1x10^{-9}$	$5x10^{-8}$
HxCDD, 1,2,3,6,7,8-	$2x10^{-9}$	$9x10^{-8}$	$1x10^{-9}$	$5x10^{-8}$	$3x10^{-9}$	$1x10^{-7}$
HxCDD, 1,2,3,7,8,9-	$1x10^{-9}$	$5x10^{-8}$	$5x10^{-10}$	$2x10^{-8}$	$2x10^{-9}$	$7x10^{-8}$
HpCDD, 1,2,3,4,6,7,8-	$3x10^{-9}$	$1x10^{-7}$	$1x10^{-9}$	$4x10^{-8}$	$4x10^{-9}$	$2x10^{-7}$
OCDD	$3x10^{-9}$	$1x10^{-7}$	$1x10^{-9}$	$4x10^{-8}$	$4x10^{-9}$	$2x10^{-7}$
TCDF, 2,3,7,8-	$1x10^{-8}$	$4x10^{-7}$	$5x10^{-9}$	$2x10^{-7}$	$2x10^{-8}$	$6x10^{-7}$
PeCDF, 1,2,3,7,8-	$4x10^{-9}$	$1x10^{-7}$	$2x10^{-9}$	$8x10^{-8}$	$6x10^{-9}$	$2x10^{-7}$
PeCDF, 2,3,4,7,8-	$7x10^{-8}$	$3x10^{-6}$	$4x10^{-8}$	$1x10^{-6}$	$1x10^{-7}$	$4x10^{-6}$
HxCDF, 1,2,3,4,7,8-	$1x10^{-7}$	$4x10^{-6}$	$5x10^{-8}$	$2x10^{-6}$	$1x10^{-7}$	$6x10^{-6}$
HxCDF, 1,2,3,6,7,8-	$2x10^{-8}$	$6x10^{-7}$	$8x10^{-9}$	$3x10^{-7}$	$2x10^{-8}$	$9x10^{-7}$
HxCDF, 1,2,3,7,8,9-	$2x10^{-9}$	$7x10^{-8}$	$9x10^{-10}$	$4x10^{-8}$	$3x10^{-9}$	$1x10^{-7}$
HxCDF, 2,3,4,6,7,8-	$5x10^{-9}$	$2x10^{-7}$	$3x10^{-9}$	$1x10^{-7}$	$8x10^{-9}$	$3x10^{-7}$
HpCDF, 1,2,3,4,6,7,8-	$2x10^{-8}$	$9x10^{-7}$	$9x10^{-9}$	$4x10^{-7}$	$3x10^{-8}$	$1x10^{-6}$
HpCDF, 1,2,3,4,7,8,9-	$5x10^{-10}$	$2x10^{-8}$	$2x10^{-10}$	$9x10^{-9}$	$7x10^{-10}$	$3x10^{-8}$
OCDF	$2x10^{-9}$	$9x10^{-8}$	$6x10^{-10}$	$3x10^{-8}$	$3x10^{-9}$	$1x10^{-7}$

Table 3-13. Hypothetical Cancer Risks Associated with Consumption of Blue Crab and Striped Bass

Chemicals of Potential Concern	Blue Crab Risks		Striped Bass Risks		Total Risks	
	Typical	RME (a)	Typical	RME (a)	Typical	RME (a)
PAHs						
Benzo(a)anthracene	$2x10^{-10}$	$7x10^{-9}$	$1x10^{-11}$	$4x10^{-10}$	$2x10^{-10}$	$7x10^{-9}$
Benzo(a)pyrene	$2x10^{-9}$	$7x10^{-8}$	$1x10^{-10}$	$4x10^{-9}$	$2x10^{-9}$	$7x10^{-8}$
Benzo(b)fluoranthene	$2x10^{-10}$	$7x10^{-9}$	$1x10^{-11}$	$4x10^{-10}$	$2x10^{-10}$	$7x10^{-9}$
Benzo(k)fluoranthene	$2x10^{-11}$	$7x10^{-10}$	$1x10^{-12}$	$4x10^{-11}$	$2x10^{-11}$	$7x10^{-10}$
Chrysene	$2x10^{-12}$	$8x10^{-11}$	$1x10^{-13}$	$5x10^{-12}$	$2x10^{-12}$	$8x10^{-11}$
Dibenzo(a,h)anthracene	$4x10^{-10}$	$2x10^{-8}$	$1x10^{-11}$	$4x10^{-10}$	$4x10^{-10}$	$2x10^{-8}$
Indeno(1,2,3-c,d)pyrene	$9x10^{-11}$	$4x10^{-9}$	$3x10^{-12}$	$1x10^{-10}$	$1x10^{-10}$	$4x10^{-9}$
PCB Coplanars						
TetraCB, 3,3',4,4'- (IUPAC #77)	$2x10^{-7}$	$8x10^{-6}$	$1x10^{-7}$	$4x10^{-6}$	$3x10^{-7}$	$1x10^{-5}$
PentaCB, 2',3,4,4',5- (IUPAC #123)	$1x10^{-8}$	$5x10^{-7}$	$7x10^{-9}$	$3x10^{-7}$	$2x10^{-8}$	$8x10^{-7}$
PentaCB, 2,3',4,4',5- (IUPAC #118)	$1x10^{-7}$	$5x10^{-6}$	$7x10^{-8}$	$3x10^{-6}$	$2x10^{-7}$	$8x10^{-6}$
PentaCB, 2,3,3',4,4'- (IUPAC #105)	$7x10^{-8}$	$3x10^{-6}$	$4x10^{-8}$	$2x10^{-6}$	$1x10^{-7}$	$4x10^{-6}$
PentaCB, 2,3,4,4',5- (IUPAC #114)	$4x10^{-9}$	$2x10^{-7}$	$2x10^{-9}$	$9x10^{-8}$	$7x10^{-9}$	$3x10^{-7}$
PentaCB, 3,3',4,4',5- (IUPAC #126)	$8x10^{-8}$	$3x10^{-6}$	$4x10^{-8}$	$2x10^{-6}$	$1x10^{-7}$	$5x10^{-6}$
HexaCB, 2,3',4,4',5,5'- (IUPAC #167)	$1x10^{-8}$	$6x10^{-7}$	$8x10^{-9}$	$3x10^{-7}$	$2x10^{-8}$	$9x10^{-7}$
HexaCB, 2,3,3',4,4',5'- (IUPAC #157)	$3x10^{-9}$	$1x10^{-7}$	$2x10^{-9}$	$7x10^{-8}$	$5x10^{-9}$	$2x10^{-7}$
HexaCB, 2,3,3',4,4',5- (IUPAC #156)	$1x10^{-8}$	$4x10^{-7}$	$5x10^{-9}$	$2x10^{-7}$	$2x10^{-8}$	$6x10^{-7}$
HexaCB, 3,3',4,4',5,5'- (IUPAC #169)	$2x10^{-9}$	$7x10^{-8}$	$8x10^{-10}$	$3x10^{-8}$	$3x10^{-9}$	$1x10^{-7}$
HeptaCB, 2,3,3',4,4',5,5'- (IUPAC #189)	$3x10^{-9}$	$1x10^{-7}$	$1x10^{-9}$	$5x10^{-8}$	$4x10^{-9}$	$2x10^{-7}$

Table 3-13. Hypothetical Cancer Risks Associated with Consumption of Blue Crab and Striped Bass

Chemicals of Potential Concern	Blue Crab Risks		Striped Bass Risks		Total Risks	
	Typical	RME (a)	Typical	RME (a)	Typical	RME (a)
Pesticides						
Aldrin	$9x10^{-9}$	$4x10^{-7}$	$5x10^{-9}$	$2x10^{-7}$	$1x10^{-8}$	$6x10^{-7}$
alpha-Chlordane	$1x10^{-9}$	$5x10^{-8}$	$7x10^{-10}$	$3x10^{-8}$	$2x10^{-9}$	$8x10^{-8}$
beta-BHC	$3x10^{-10}$	$1x10^{-8}$	$1x10^{-10}$	$5x10^{-9}$	$4x10^{-10}$	$2x10^{-8}$
DDD, 4,4'-	$2x10^{-9}$	$7x10^{-8}$	$9x10^{-10}$	$4x10^{-8}$	$3x10^{-9}$	$1x10^{-7}$
DDE, 4,4'-	$6x10^{-10}$	$3x10^{-8}$	$3x10^{-10}$	$1x10^{-8}$	$1x10^{-9}$	$4x10^{-8}$
DDT, 4,4'-	$7x10^{-10}$	$3x10^{-8}$	$4x10^{-10}$	$2x10^{-8}$	$1x10^{-9}$	$5x10^{-8}$
Dieldrin	$6x10^{-9}$	$2x10^{-7}$	$2x10^{-9}$	$9x10^{-8}$	$9x10^{-9}$	$3x10^{-7}$
gamma-Chlordane	$2x10^{-9}$	$6x10^{-8}$	$8x10^{-10}$	$3x10^{-8}$	$2x10^{-9}$	$1x10^{-7}$
Semivolatiles						
Bis(2-ethylhexyl)phthalate	$3x10^{-11}$	$1x10^{-9}$	$2x10^{-12}$	$7x10^{-11}$	$3x10^{-11}$	$1x10^{-9}$
Total	$3x10^{-6}$	$1x10^{-4}$	$4x10^{-6}$	$2x10^{-4}$	$7x10^{-6}$	$3x10^{-4}$

a. RME = Reasonable Maximum Exposure

Table 3-14. Percent Contribution of CPC to Cancer Risks Associated with Consumption of Blue Crab and Striped Bass

Chemicals of Potential Concern	Total Risk		Percent Contribution to Total Risk (a)		Cumulative Percent Contribution	
	Typical	RME (b)	Typical	RME (b)	Typical	RME (b)
Arsenic	$4x10^{-6}$	$2x10^{-4}$	62	62	62	62
Beryllium	$9x10^{-7}$	$4x10^{-5}$	12	12	74	74
TCDD, 2,3,7,8-	$7x10^{-7}$	$3x10^{-5}$	9.0	9.0	83	83
TetraCB, 3,3',4,4'- (IUPAC #77)	$3x10^{-7}$	$1x10^{-5}$	4.3	4.3	87	87
PentaCB, 2,3',4,4',5- (IUPAC #118)	$2x10^{-7}$	$8x10^{-6}$	2.9	2.9	90	90
HxCDF, 1,2,3,4,7,8-	$1x10^{-7}$	$6x10^{-6}$	2.0	2.0	92	92
PentaCB, 3,3',4,4',5- (IUPAC #126)	$1x10^{-7}$	$5x10^{-6}$	1.7	1.7	94	94
PentaCB, 2,3,3',4,4'- (IUPAC #105)	$1x10^{-7}$	$4x10^{-6}$	1.5	1.5	96	96
PeCDF, 2,3,4,7,8-	$1x10^{-7}$	$4x10^{-6}$	1.4	1.4	97	97
HpCDF, 1,2,3,4,6,7,8-	$3x10^{-8}$	$1x10^{-6}$	0.42	0.42	97	97
HxCDF, 1,2,3,6,7,8-	$2x10^{-8}$	$9x10^{-7}$	0.32	0.32	98	98
HexaCB, 2,3',4,4',5,5'- (IUPAC #167)	$2x10^{-8}$	$9x10^{-7}$	0.31	0.31	98	98
PentaCB, 2',3,4,4',5- (IUPAC #123)	$2x10^{-8}$	$8x10^{-7}$	0.29	0.29	98	98
HexaCB, 2,3,3',4,4',5- (IUPAC #156)	$2x10^{-8}$	$6x10^{-7}$	0.22	0.22	99	99
TCDF, 2,3,7,8-	$2x10^{-8}$	$6x10^{-7}$	0.21	0.21	99	99
Aldrin	$1x10^{-8}$	$6x10^{-7}$	0.19	0.19	99	99
Dieldrin	$9x10^{-9}$	$3x10^{-7}$	0.12	0.12	99	99
HxCDF, 2,3,4,6,7,8-	$8x10^{-9}$	$3x10^{-7}$	0.11	0.11	99	99
PentaCB, 2,3,4,4',5- (IUPAC #114)	$7x10^{-9}$	$3x10^{-7}$	0.090	0.090	99	99
PeCDD, 1,2,3,7,8-	$6x10^{-9}$	$2x10^{-7}$	0.081	0.080	99	99
PeCDF, 1,2,3,7,8-	$6x10^{-9}$	$2x10^{-7}$	0.076	0.076	99	99
HexaCB, 2,3,3',4,4',5'- (IUPAC #157)	$5x10^{-9}$	$2x10^{-7}$	0.065	0.065	99	99
OCDD	$4x10^{-9}$	$2x10^{-7}$	0.060	0.060	100	100
Total	$7x10^{-6}$	$3x10^{-4}$	>99	>99		

a. Based on calculations prior to rounding of total risk numbers
b. RME = Reasonable Maximum Exposure

3.5.2 Noncarcinogenic Hazard

Noncarcinogenic hazard indices were estimated for human exposure to bioaccumulative chemicals from ingestion of striped bass and blue crab. Hazard indices were calculated by dividing exposures, expressed as the ADIs calculated in Section 3.3.3, by the appropriate reference doses, as reported in Section 3.4. The equation for estimating the noncarcinogenic hazard quotient is as follows:

Hazard Quotient = ADI / RfD

where:

Hazard Quotient	=	Ratio of estimated doses to toxicity criteria;
ADI	=	Average daily intake (mg/kg-day); and
RfD	=	Reference Dose (mg/kg-day).

Composite hazard indices were then calculated by summing across all chemicals, without regard to target organ, to initially determine the total hazard index for individuals consuming striped bass and blue crab. The chemical-specific noncarcinogenic hazard quotients for typical and RME exposures resulting from the consumption of striped bass and blue crab are reported in Table 3-15. The total hazard index for combined consumption of striped bass and blue crab is estimated to be 0.27 for typical exposure and 3.3 for the RME. A hazard index greater than 1 is considered by EPA to represent a potential concern for noncarcinogenic health effects (EPA, 1989a, 1995).

Chemicals contributing 1% or greater of the total hazard index, in order of contribution are: arsenic (24%); monobutyltin (12%); antimony (12%); Aroclor 1248 (8.8%); aluminum (7.0%); mercury (6.2%); dibutyltin (5.4%); thallium (4.3%); cadmium (3.5%); copper (3.4%); vanadium (2.9%); Aroclor 1254 (2.1%); barium (1.6%); nickel (1.6%); manganese (1.5%); and zinc (1.0%). Collectively, these chemicals represent 98 percent of the total hazard index estimated in this assessment (Table 3-16).

As shown in Table 3-17, the group of inorganic chemicals contributes the greatest proportion (74 %) of the total risk for cancer, followed, in descending order, by PCDD/Fs (14%) and coplanar PCBs (12%). For noncarcinogenic hazard, inorganic chemicals contribute the greatest proportion

Table 3-15. Noncancer Hazard Indices Associated with Consumption of Blue Crab and Striped Bass

Chemicals of Potential Concern	Blue Crab Hazard Quotient		Striped Bass Hazard Quotient		Total Hazard Quotient	
	Typical	RME (a)	Typical	RME (a)	Typical	RME (a)
PAHs						
Acenaphthene	1.4×10^{-8}	1.6×10^{-7}	3.8×10^{-10}	4.6×10^{-9}	1.4×10^{-8}	1.7×10^{-7}
Acenaphthylene	2.0×10^{-8}	2.4×10^{-7}	5.9×10^{-10}	7.1×10^{-9}	2.1×10^{-8}	2.5×10^{-7}
Anthracene	3.4×10^{-9}	4.1×10^{-8}	1.2×10^{-10}	1.4×10^{-9}	3.6×10^{-9}	4.2×10^{-8}
Benzo(ghi)perylene	3.0×10^{-8}	3.6×10^{-7}	1.0×10^{-9}	1.2×10^{-8}	3.1×10^{-8}	3.7×10^{-7}
Dibenzofuran	1.8×10^{-7}	2.2×10^{-6}	5.1×10^{-9}	6.2×10^{-8}	1.9×10^{-7}	2.2×10^{-6}
Fluoranthene	9.8×10^{-8}	1.2×10^{-6}	5.5×10^{-9}	6.6×10^{-8}	1.0×10^{-7}	1.2×10^{-6}
Fluorene	2.0×10^{-8}	2.4×10^{-7}	6.0×10^{-10}	7.2×10^{-9}	2.1×10^{-8}	2.5×10^{-7}
Phenanthrene	8.2×10^{-8}	9.7×10^{-7}	2.9×10^{-9}	3.5×10^{-8}	8.5×10^{-8}	1.0×10^{-6}
Pyrene	1.2×10^{-7}	1.4×10^{-6}	6.8×10^{-9}	8.2×10^{-8}	1.3×10^{-7}	1.5×10^{-6}
PCBs						
Aroclor 1248	0.016	0.19	0.0084	0.10	0.024	0.29
Aroclor 1254	0.0038	0.045	0.0020	0.024	0.0058	0.070
Pesticides						
Aldrin	0.00014	0.0017	7.5×10^{-5}	0.00091	0.00014	0.0026
alpha-Chlordane	0.00013	0.0016	7.2×10^{-5}	0.00087	0.00013	0.0024
DDT, 4,4'-	3.4×10^{-5}	0.00040	1.8×10^{-5}	0.00022	5.2×10^{-5}	0.00062
delta-BHC	4.8×10^{-6}	5.6×10^{-5}	2.3×10^{-6}	2.8×10^{-5}	7.1×10^{-6}	8.4×10^{-5}
Dieldrin	6.0×10^{-5}	0.00071	2.2×10^{-5}	0.00027	8.3×10^{-5}	0.00098
Endrin	3.5×10^{-5}	0.00042	1.9×10^{-5}	0.00022	5.4×10^{-5}	0.00064
gamma-Chlordane	0.00015	0.0018	8.5×10^{-5}	0.0010	0.00015	0.0028
Methoxychlor	3.9×10^{-6}	4.6×10^{-5}	2.1×10^{-6}	2.5×10^{-5}	6.0×10^{-6}	7.1×10^{-5}

Table 3-15. Noncancer Hazard Indices Associated with Consumption of Blue Crab and Striped Bass

Chemicals of Potential Concern	Blue Crab Hazard Quotient		Striped Bass Hazard Quotient		Total Hazard Quotient	
	Typical	RME (a)	Typical	RME (a)	Typical	RME (a)
Inorganics						
Aluminum	0.0069	0.081	0.012	0.15	0.019	0.23
Antimony	0.012	0.14	0.021	0.25	0.033	0.39
Arsenic	0.024	0.28	0.042	0.51	0.066	0.79
Barium	0.0015	0.018	0.0027	0.033	0.0043	0.051
Beryllium	0.00012	0.0014	0.00021	0.0025	0.00032	0.0039
Cadmium	0.0034	0.040	0.0060	0.073	0.0094	0.11
Chromium	0.00068	0.0080	0.0012	0.015	0.0019	0.023
Cobalt	0.00012	0.0014	0.00021	0.0025	0.00033	0.0039
Copper	0.0033	0.039	0.0059	0.071	0.0092	0.11
Manganese	0.0014	0.017	0.0026	0.031	0.0040	0.048
Mercury	0.0061	0.072	0.011	0.13	0.017	0.20
Nickel	0.0015	0.018	0.0027	0.033	0.0043	0.051
Selenium	0.00015	0.0018	0.00027	0.0032	0.00042	0.0050
Silver	0.00067	0.0079	0.0012	0.014	0.0019	0.022
Thallium	0.0042	0.050	0.0075	0.091	0.012	0.14
Vanadium	0.0029	0.034	0.0051	0.062	0.0080	0.096
Zinc	0.00099	0.012	0.0017	0.021	0.0027	0.033
Semivolatiles						
Bis(2-ethylhexyl)phthalate	8.6×10^{-7}	1.0×10^{-5}	4.6×10^{-8}	5.5×10^{-7}	9.0×10^{-7}	1.1×10^{-5}
Butyl benzyl phthalate	3.2×10^{-9}	3.8×10^{-8}	1.9×10^{-10}	2.3×10^{-9}	3.4×10^{-9}	4.0×10^{-8}
Di-n-butyl phthalate	6.9×10^{-9}	8.1×10^{-8}	4.1×10^{-10}	4.9×10^{-9}	7.3×10^{-9}	8.6×10^{-8}
Di-n-octyl phthalate	4.3×10^{-8}	5.1×10^{-7}	2.7×10^{-9}	3.2×10^{-8}	4.6×10^{-8}	5.4×10^{-7}
Trichlorobenzene, 1,2,4-	7.1×10^{-8}	8.4×10^{-7}	2.2×10^{-9}	2.6×10^{-8}	7.3×10^{-8}	8.7×10^{-7}
Miscellaneous						
Dibutyltin	0.0053	0.062	0.0093	0.11	0.015	0.17
Monobutyltin	0.0074	0.087	0.026	0.32	0.034	0.40
Total	0.10	1.2	0.17	2.1	0.27	3.3

a. RME = Reasonable Maximum Exposure

Table 3-16. Percent Contribution of CPC to Noncancer Hazard Indices Associated with Consumption of Blue Crab and Striped Bass

Chemicals of Potential Concern	Total Hazard Quotient		Percent Contribution to Total Hazard (a)		Cummulative Percent Contribution	
	Typical	RME (b)	Typical	RME (b)	Typical	RME (b)
Arsenic	0.066	0.79	24	24	24	24
Monobutyltin	0.034	0.40	12	12	37	37
Antimony	0.033	0.39	12	12	49	49
Aroclor 1248	0.024	0.29	8.8	8.8	57	57
Aluminum	0.019	0.23	7.0	7.0	64	64
Mercury	0.017	0.20	6.2	6.2	71	71
Dibutyltin	0.015	0.17	5.4	5.4	76	76
Thallium	0.012	0.14	4.3	4.3	80	80
Cadmium	0.0094	0.11	3.5	3.5	84	84
Copper	0.0092	0.11	3.4	3.4	87	87
Vanadium	0.0080	0.10	2.9	2.9	90	90
Aroclor 1254	0.0058	0.070	2.1	2.1	92	92
Barium	0.0043	0.051	1.6	1.6	94	94
Nickel	0.0043	0.051	1.6	1.6	95	95
Manganese	0.0040	0.048	1.5	1.5	97	97
Zinc	0.0027	0.033	1.0	1.0	98	98
Total	0.27	3.2	>97	>97		

a. Based on calculations prior to rounding of total hazard quotient
b. RME = Reasonable Maximum Exposure

Table 3-17. Total Carcinogenic Risks and Noncarcinogenic Hazards at the Site

Chemical Groups	Total Carcinogenic Risk(a) Typical	RME (b)	Percent Contribution to Typical Risk	Percent Contribution to RME Risk
Total Inorganics	$5x10^{-6}$	$2x10^{-4}$	74	74
Total PCDD/Fs	$1x10^{-6}$	$4x10^{-5}$	14	14
Total Coplanar PCBs	$8x10^{-7}$	$3x10^{-5}$	12	11
Total Pesticides	$3x10^{-8}$	$1x10^{-6}$	0.45	0.44
Total PAHs	$3x10^{-9}$	$1x10^{-7}$	0.038	0.038
Total Semivolatiles	$3x10^{-11}$	$1x10^{-9}$	0.00045	0.00044
Total Risk	$7x10^{-6}$	$3x10^{-4}$	100	100

	Total Noncarcinogenic Hazard (a) Typical	RME (b)	Percent Contribution to Typical Hazard Ratio	Percent Contribution to RME Hazard Ratio
Total Inorganics	0.19	2.3	71	71
Total Butyltins	0.048	0.58	18	18
Total Aroclors	0.030	0.36	11	11
Total Pesticides	0.00086	0.010	0.32	0.31
Total Semivolatiles	$1.0x10^{-6}$	$1.2x10^{-5}$	0.00038	0.00037
Total PAHs	$6.0x10^{-7}$	$7.1x10^{-6}$	0.00022	0.00022
Total Hazard Quotient	0.27	3.3	100	100

a. Combined risk/hazard from consumption of blue crab and striped bass.
b. RME = Reasonable Maximum Exposure.

of the hazard (71%) followed, in descending order, by butyltins (18%) and Aroclor mixtures of PCBs (11%).

3.5.3 Identification of Uncertainties

An important facet of the method and use of human health risk assessment concerns the recognition of uncertainties and limitations inherent in the process which arise in connection with dose-response models, animal to human extrapolation, chemical fate and transport, models of potential exposure, and site-specific characteristics. From a regulatory perspective, these uncertainties and limitations may be addressed by developing and employing assumptions which typically overestimate the magnitude of many variables. In this fashion, agencies charged with the protection of public health have often assumed that their mandate would best be met by overestimating potential risks from exposure to environmental contaminants (Paustenbach, 1990b). However, as our awareness of these uncertainties improves, along with our understanding of how to best characterize them, the result will almost certainly be risk assessments that are more credible and thus more useful to risk managers (Paustenbach, 1990b; Keenan et al., 1994). To that end, recent EPA risk assessment guidance (EPA, 1992) incorporates refinements in the treatment of uncertainty. Following are discussions of the major sources of uncertainty associated with the present assessment.

Presumed Additivity of Dioxin Congeners

For the purposes of this HHRA, it has been assumed that the biological activities of the individual dioxin congeners may be summed to develop an aggregate dioxin risk (i.e., the I-TEF scheme). However, there is recent evidence demonstrating that the biological activity of PCDD/F congeners in environmental media may be far less than additive. Specifically, the expected biological activity of the PCDD/F mixture may be much greater than measured in an *in vitro* or *in vivo* assay system. For example, Safe et al. (1989) examined the biological activity of a municipal fly ash extract which contained numerous PCDD/Fs. The 2,3,7,8- equivalence of the extract, obtained via Gas Chromatograph/Mass Spectrometer (GC/MS) analysis, was 9,350 ppt. However, the *in vitro* activity of the extract, as measured by aryl hydrocarbon hydroxylase (AHH) induction, was only 105 ppt. Even more significantly, the *in vivo* activity of the extract, as measured by AHH induction, was only 75 ppt. As noted by the authors:

> "the 'toxic equivalents' of these PCDD and PCDF-containing extracts are significantly lower than the total concentrations of these compounds."

A review of the literature indicates that, even when reconstituted mixtures of PCDD/Fs are evaluated (i.e., mixtures prepared by the investigator in the laboratory, similar discrepancies between "estimated" vs. "measured" 2,3,7,8-TCDD TEF are noted. For example, Nagao et al. (1993) showed that the potency of two different PCDF mixtures (as measured by induction of cleft palate in mice), was significantly less than expected. As noted by the authors:

> "With respect to the PCDF mixtures, the use of TEF factors...has a tendency to overestimate the real potency."

In no case has anybody shown greater than additive effects, and even close-to-additive effects can only be produced with carefully controlled reconstituted mixtures. Some have suggested that the reduced activity of complex PCDD/F mixtures is due to the large excess of congeners with weak or no biological activity, which interferes with the potency of the main toxic components (Neubert et al., 1992).

In summary, the "chemical-derived TEF" may far overestimate the true "biological TEF" to the extent that, based on chemical evidence alone, the presence of a dioxin risk may be presumed when in fact none exists. This would be particularly true for environmental media which contain a relatively high proportion of the least active congeners (e.g., OCDD/F), such as the sediments it the Site. For this reason, the presumption of additivity of the PCDD/F TEFs in this HHRA may overestimate the true extent of the PCDD/F related risks at the Site.

Reference Doses and Hazard Quotient Estimates

Significant uncertainty is associated with the evaluation of noncarcinogenic effects of chemicals in the environment. Primary sources of uncertainty include the derivation and use of chemical-specific toxicity values and the limitations inherent in the hazard index methodology, such as the assumption of additivity for multiple chemical exposure and the inability of the Hazard Quotient (HQ) to predict the likelihood of adverse effects occurring at doses above the RfD.

Toxicity values based on human epidemiological studies are not available for most chemicals, and in general human studies suffer from a lack of exposure data and any number of potential confounding factors, including concomitant exposure to multiple chemicals, recall bias, and lifestyle effects. Therefore, for many chemicals, data from studies of laboratory animals provide the basis for toxicity values. The practice of extrapolating effects observed in experimental animals to predict human toxic response to chemicals is a major source of uncertainty in risk assessment (EPA, 1989a).

A hazard quotient (HQ) is the ratio of the estimated chronic intake level of a chemical to the reference dose (RfD) for that chemical (EPA, 1989a). Since the RfD is established at a dose level at and below which adverse effects would not be expected, an HQ at or below 1 is considered to be a level that would not result in an increased health risk (EPA, 1989a). Given that many environmental contamination situations involve exposure to more than one chemical, the HQs for the individual chemicals may be summed to determine an Hazard Index (HI) for the mixture. Therefore, a HI is typically defined as the sum of HQs for the individual chemicals of concern at the site. This approach assumes that exposures to multiple chemicals may result in adverse effects even if no single chemical exposure exceeds its RfD. As with single contaminant exposures, an HI at or below 1.0 is regarded as unlikely to result in an increased health risk even for sensitive populations (EPA, 1989a).

EPA (1989a) guidance, specifying that individual HQs and total site HIs should not exceed a value of 1, represents conservative and health protective regulatory toxicological criteria. That is, a HI value greater than 1 does not necessarily indicate that adverse health effects are likely, because the RfD contains a measure of conservatism to ensure health protection.

First, the development of RfDs is a highly conservative process. RfDs are generally developed by dividing NOAELs from animal studies by "safety factors", to adjust for uncertainties in the physiological differences between humans and laboratory animals, variation in sensitivity among individuals of human subpopulations, and differences between subchronic and chronic exposures. These ten-fold safety factors are typically applied in multiples of 10 to NOAELs. Thus, when all three factors are combined, the resultant safety factor is equal to 1,000 (10 x 10 x 10) (Barnes and Dourson, 1988).

However, analysis of toxicological data indicate that a value less than ten for an individual safety factor may be adequate, depending on the relative magnitude of uncertainty associated with the critical study. For example, Lewis, et al. (1990) reviewed the data from eighteen laboratory animal studies and found that the average difference between NOAELs based on subchronic exposures and NOAELs based on chronic exposures was a factor of 3.5 or less, not the default value of 10 that is typically applied. Similarly, a factor of 1 for extrapolation from laboratory animals to humans is appropriate if there are adequate data which indicate a likelihood that the test species is significantly more sensitive to the chemical-specific effect than humans.

In cases when the RfD is based on a study which reports a LOAEL but does not report a NOAEL, an additional safety factor is generally applied to the LOAEL to derive an estimated NOAEL. This safety factor may range from 1 to 10, depending upon the study and the severity of the effects observed. When Dourson and Starra (1983) compared LOAELs and NOAELs from a variety of studies that reported both, they found that 96 percent of those studies had LOAEL:NOAEL ratios of 5:1 or less. Based on their evaluation, Dourson and Starra (1983) concluded that a safety factor in the range of 1 to 10 is supportable for extrapolating from a LOAEL to a NOAEL. In addition, Dourson and Starra (1983) suggested that the severity of the effect is a critical determinant in establishing a LOAEL to NOAEL safety factor. For example, for liver necrosis, a relatively severe effect, a relatively high value (i.e., 10) was suggested. However, for a less severe effect, such as fatty infiltration of the liver, which results in increased liver weight, a factor of 3 was suggested (Dourson and Starra, 1983).

There is regulatory precedent for use of safety factors totalling less than 1,000. In calculating an RfD for 2,4-dichlorophenol, EPA applied an uncertainty (or safety) factor of 100 to the value reported as a NOAEL to account for extrapolation from animal data to humans and for protection of sensitive populations. In deriving the RfD for Aroclor 1254, the EPA applied a safety factor of 300 to the LOAEL observed in the critical study. EPA justified the safety factor of 300 by reasoning that: a 10-fold factor for interspecies was unnecessary due to similarities between humans and monkeys; only a "partial factor" was needed to account for use of a LOAEL because the effect (nail bed changes) was not considered serious; and a "reduced" factor for extrapolation from subchronic to chronic exposure was adequate because the critical effects did not appear to be

dependent upon the duration of the study. Thus, the uncertainty factor of 300 applied by EPA in this case was significantly lower than the safety factor of 10,000 which would have resulted if four individual uncertainty factors of 10 had been combined.

In conclusion, many conservative assumptions are used to account for various sources of uncertainty associated with the evaluation of noncarcinogenic effects. One example of this conservatism and the health-protective nature of HIs calculated in this assessment is the use of multiple safety factors in the derivation of the RfD. Typically, a safety factor of 1,000 is applied to the NOAEL in deriving an RfD; however, the EPA has applied combined safety factors as low as 100. Therefore, use of a safety factor of 1,000 may be overly conservative for some chemicals by a factor of ten or more (Lewis, et al., 1990).

Cancer Slope Factor and Risk Estimates

In establishing slope factors, regulatory agencies implement methods that introduce multiple sources of uncertainty that ultimately increase the overall conservatism inherent to the cancer risk estimates. Major uncertainties exist in the extrapolation from animals to humans and from high doses to low doses (51 FR 185:33992-34003, September 24, 1986). For example, species differ substantially in their uptake, metabolism, organ distribution, and target-site susceptibility of carcinogens. While laboratory animals are exposed to controlled concentrations at extremely high doses, humans are typically exposed to lower environmental levels (Crump et al., 1989). In addition, the potency of a chemical is influenced by the size and lifespan of the species experimentally exposed. This has important implications due to the long latency period of many carcinogenic responses. An individual's susceptibility to a carcinogenic compound is also influenced by the variability that exists within human populations. Variables include genetic constitution, diet, occupational and home environments, activity patterns, and other cultural factors (51 CFR 185:33992-34003, September 24, 1986).

To compensate for these various sources of uncertainty in the dose response assessment, conservatism is incorporated into the derivation of the slope factor. The slope factor represents the upper 95th percent confidence limit on the probability of a carcinogenic response per unit intake of a chemical over a lifetime (EPA, 1989c). In other words, there is only a five percent chance that the probability of a response would be greater than the estimated value. Therefore, slope factors, by definition, overestimate the actual potency of a carcinogen. The accuracy of risk estimates,

associated with low doses, predicted by the LMS model is unknown, but may in fact be zero (EPA, 1986).

Use of Relative Toxicity Values

As described by EPA (1989a), there is significant uncertainty associated with the use of relative toxicity values, such as TEFs; these uncertainties are the focus of a number of current research programs. In the absence of chemical-specific toxicity information and consistent with EPA (1989d, 1993c, 1994b) interim guidance and practice, relative toxicity schemes were employed for evaluating risks associated with exposure to PCDD/PCDFs, PAHs, and coplanar PCBs (EPA, 1989d, 1993c, 1994b). Also, as noted in Section 3.4, the inorganic arsenic oral slope factor has been applied to the total arsenic levels estimated in fish and crab tissues in order to derive an arsenic-related cancer risk for humans. The true percent composition of the total arsenic as inorganic arsenic in these tissues is unknown.

Additivity of Risk and Hazard

A high level of uncertainty is also associated with exposures to multiple chemicals. For evaluation of cumulative effects from exposure to multiple chemicals, EPA (1989a) recommends that risks be summed across chemicals for each exposure pathway. This assumption does not account for dissimilarities in mechanisms of action or synergistic or antagonistic effects, but is considered appropriate for screening levels analyses (EPA, 1992). Therefore, in this assessment it was assumed that risks and hazards are additive.

Selection of Exposure Pathways

There is considerable uncertainty regarding the extent and likelihood of exposure to chemicals in fish and crabs. Factors which would influence whether significant exposures via consumption of fish and crabs might actually occur include aesthetic factors such as the desirability of fishing at the Site, access to the River and the availability and abundance of edible species at the Site. Although it is difficult to determine the impact of these factors on exposure, for this screening-level assessment, it was assumed that potential exposure via fish and crab consumption is a plausible exposure pathway.

Selection of Chemicals of Potential Concern

Uncertainty in the risk assessment arises during the selection of CPC. A limited number of organic chemicals in sediment were screened out from quantitative evaluation because they are not considered bioaccumulative. Thus, their contribution to site-wide health risks was considered to be negligible. Because these chemicals were not selected as CPC, potential cancer and noncancer risks associated with their presence at the site were not included in the quantitative risk assessment, and therefore contribute to the overall uncertainty of the risk estimates.

Exposure Point Concentrations

In the absence of data on chemical concentrations in edible biota (i.e., blue crabs and striped bass), concentrations were estimated from 95% upper confidence limits on the mean surface sediment concentrations at the Site using a food web model or empirical relationships, as described in Section 4.4. Similar to direct evaluation techniques, there is a degree of uncertainty associated with the final tissue concentrations derived from use of the food web model or empirical relationships. Although all efforts were made to estimate chemical accumulation in biota as accurately as possible, several uncertainties are associated with such estimations in the absence of Site-specific data. The uncertainties associated with the use of the food web model are discussed in detail in Section 4.6.3.

As discussed in Section 3.3.1, estimated concentrations of chemicals in muscle tissue were used as exposure point concentrations in blue crab. This assumption is appropriate, since the vast majority of anglers do not eat the remainder of crabs (other than the edible muscle tissue), including the hepatopancreas. A study by Landolt et al. (1985) evaluated the consumption of recreationally caught crab from the Puget Sound area and reported that only 0 to 0.8 percent of all anglers consume the crab hepatopancreas. Thus, greater than 99 percent of the population of anglers eat only the muscle tissue of crabs. For striped bass, it was assumed that anglers did not consume the whole fish, but instead consume only the fillets. The assumption that individuals do not eat the whole body of recreationally caught fish is supported by several studies (Ebert et al., 1993; EPA, 1989b).

To determine a fillet contaminant tissue concentration for organic chemicals in striped bass, it was assumed that the edible portion contained 2.28 percent lipid. This value represents the mean concentration of lipid measured in edible fillets of striped bass from the lower Passaic River

(Belton et al., 1985; Hauge et al., 1990, 1993). The value of 2.28 percent is Site-specific, and therefore, should allow the close approximation of a fillet contaminant tissue concentration. Similarly, a Site-specific value for percent lipids in the muscle tissue of blue crab (0.78 %) was used, based on data from Belton et al. (1985) and Hauge et al. (1990, 1993).

As discussed in Section 3.3.1, few quantitative data are available regarding the bioaccumulation of inorganic chemicals in aquatic organisms. For chromium and lead, data from a study in aquatic organisms from the Hackensack River by Hall and Pullian (1995) were used to estimate ratios of biota-to-sediment metal concentrations (K_s). In this study, the authors derived K_s for blue crab of 0.02 and 0.1 for chromium and lead, respectively. As described in Section 4.4.2.4, K_s values of 0.01 and 0.05 were used for chromium and lead in striped bass, respectively, reflecting an assumption that migratory striped bass are only present at the Site for a limited portion of the year. For other inorganic chemicals, a K_s of 0.5 was used for blue crab, reflecting an assumption that 50% of the chemical concentration in sediments would be found in the whole body of blue crabs. This estimate was based on gross observations of K_s for a number of metals from previous investigations in the New York/New Jersey Harbor Estuary (O'Connor and Rachlin, 1982). Similarly, a K_s of 0.25 was used for migratory striped bass, as described in Section 4.4.2.4. In addition, 30% of the whole body chemical concentrations were assumed to be available in the edible portions of fish and crab, as previously discussed.

Another important uncertainty associated with the exposure point concentrations is the assumption that chemical concentrations in fish tissue will remain constant over the entire exposure period. It is likely that fish tissue levels will decrease over time as sediment concentrations decrease or as contaminated sediments in the BAZ are naturally buried. On the other hand, certain contaminant levels may increase due to ongoing releases.

Cooking Loss

The risk assessment for the Site did not consider a reduction in the concentration of organic chemicals in fish after cooking. This reduction is attributable to separation of contaminated lipid from the remaining fish tissue during cooking. In addition, volatilization may account for added losses when, under higher temperatures, the chemical is released. Loss of lipids is a function of

the temperature and cooking duration, with higher temperatures and longer cooking times causing a greater loss of fat and accumulated chemicals from the edible tissue. As a result, cooking methods such as frying, baking, or broiling are particularly effective at removing organic chemicals.

Results of several studies indicate that cooking can lead to substantial reductions in organic chemical concentrations in fish tissues. For example, the results of Sherer and Price (1993), indicate that cooking leads to reductions in PCB levels in fish ranging from 0 to 74 percent. Similarly, studies by Stachiw et al. (1988), and Zabik and Zabik (1995), have shown reductions in TCDD concentrations ranging from about 30 to 100 percent. Finally, several studies have shown that cooking can reduce pesticide concentrations 2 to 72 percent in fish tissue (Reinert et al., 1972; Smith et al., 1973; Zabik et al., 1982). For these reasons, regulatory agencies frequently recommend that anglers cook their fish before consumption and that they use a cooking method that does not reuse the fish oils (NYSDEC, 1991).

Because the actual dose received by anglers during consumption is determined by the amount of chemical in each fish meal, any reduction that occurs during the cooking process will result in a reduction in the exposure concentration. Research has shown that anglers in the northeastern United States typically use cooking methods that reduce organochlorine levels in self-caught fish, with frying, baking and broiling 62, 18, and 16 percent of the time, respectively (ChemRisk, 1992; Connelly et al., 1992). As a result, the exclusion of a factor for cooking loss in the risk assessment is likely to lead to an overestimation in the actual chemical concentration consumed by recreational anglers.

Fish Consumption Rate

There is uncertainty associated with the fish consumption rates employed in this analysis. As discussed in detail in Section 3.3.2, in the absence of site-specific data, the fish consumption rates used in this assessment were selected from the best available studies that are representative of the physical and demographic characteristics of the Site. Actual consumption of fish from the Site may be substantially lower than the rates reported in the Price et al. (1994) reanalysis of the Puffer et al. (1981) and Pierce et al. (1981) studies due to restricted access to the Site and lack of aesthetic appeal to recreational anglers and low productivity of the fishery.

Exposure Duration

As recommended by EPA (1989a) a 30-year exposure period was assumed for the RME. The basis for this assumption is the adage that an individual resides in the same house for 30 years. EPA has stated that this value represents the 90th percentile of the length of time a typical homeowner will live in the same house (EPA, 1989c).

Residential mobility is an accurate predictor of exposure duration for many sources of contamination that occur in or near the home. An individual's potential exposure to indoor air pollution or contaminated soil, air, and groundwater near their residence is a function of the amount of time spent at home. This exposure may conceivably continue throughout the individual's lifetime unless the person changes their residence.

However, the duration of time an individual remains in one residence may not be a reasonable predictor of the duration of angling from a particular waterbody. An individual may give up angling and not change their residence or may move to a nearby residence and keep fishing the same waterbody. Unlike other types of exposures which often result from proximity to the source, potential exposure from fishing must be actively sought and is only partially dependent on the location of an angler's current residence. Exposure from consuming recreationally caught fish will be most significant for those individuals who continue to fish the waterbody of concern regardless of their current residence. As a result other factors in addition to residential mobility must be considered when predicting the duration of exposure from fish consumption.

A critical component of any risk assessment is estimating how long or how often an individual may be exposed to the chemicals of potential concern. In the case of the tidal Passaic River, exposure duration should be defined as the time an angler begins fishing and continuing until the angler no longer catches and consumes fish from the Site. The point at which an angler stops fishing varies with the individual angler. Three factors influence the time when an angler stops fishing: 1) the probability that an individual will relocate from his/her current residence (mobility); 2) the probability that an individual will decide to no longer participate in the sport of fishing (angling cessation); and 3) the probability that an individual will die (mortality). The duration of exposure can only be properly estimated when these three factors are considered. In this screening level

HHRA, such an analysis was beyond the scope of our quantitative evaluation. However, it is instructive to discuss these uncertainties in a qualitative fashion in order to demonstrate that by not considering them, the potential risks to human health are almost certainly overstated.

Mobility

When evaluating the influence of the mobility factor on exposure duration for fish consumption, it is necessary to go beyond a strict consideration of residential mobility because, as described above, changes in household location may not lead to changes in fishing behavior. Only when an individual moves a sufficient distance will a change likely be made in preferred fishing locations. While interstate or U.S. regional mobility data could be used to estimate the number of individuals who give up fishing at a preferred fishing location (due to a significant move in distance), interstate moves (within state) that would also result in a change in angling practices also need to be considered. It is likely that the actual number of anglers who stop fishing at a specific location would be underestimated by relying on interstate or regional mobility data. County mobility, however, may be an appropriate surrogate for representing the probability that an individual gives up angling because he/she moves sufficiently far enough away. These data are available from the U.S. Bureau of Census (1988, 1991) which publishes information on the number of individuals who move out of a given county, but still remain within the same state.

Factors such as age and gender can influence mobility. For example, the frequency of moving is highly dependent on age. Individuals between the ages of 20 to 29 have a greater probability of moving than individuals over 30. Gender also has an impact on mobility. Due to gender-specific tendencies, men are somewhat more likely to move than are women (U.S. Bureau of the Census, 1991). To account for these patterns and to identify the range of variability found in the angler population, it is necessary to identify a distribution of intercounty mobility rates for males and females of each age. Specifically, data on county mobility by age group and gender in the Northeast region are appropriate.

Angling Cessation

In addition to moving, an angler may give up fishing due to lack of interest, bad weather, increasing age, or a number of other reasons. In fact, at every age there is a certain probability that

an individual will permanently give up the sport. However, due to the difficulty of collecting these data, no study has specifically evaluated this phenomenon. Not only is it difficult for individuals to predict whether they will give up fishing, individuals who report giving up fishing one year may only temporarily withdraw from the sport. These same individuals may start and stop fishing many times over the course of their lifetimes.

A survey in the State of Maine determined that 72 percent of all licensed anglers fish every year once they start fishing (Boyle et al., 1990). This study supports the fact that the majority of anglers are extremely dedicated to their sport, indicating that the number of anglers in the total state population should be relatively constant between years. This type of information can be used to determine the age-specific probability that an individual will permanently cease angling. A similar comparison of the number of anglers in New Jersey to the total state population will identify the relative number of anglers at each age. The change in the number of anglers with increasing age can then be used to estimate the probability that an individual will give up angling.

As an example, an initial analysis using data collected by ChemRisk (1992) indicates that the percentage of anglers in the population increases from age 18 until the mid-20s, where it remains relatively constant for about 20 years. In the mid-40s until the late 60s angling begins to decline significantly. Finally, after about the age of 67, the number of anglers is again roughly stable until age 81, the oldest age recorded in the survey. A similar type of analysis could be performed using New Jersey data if available.

Mortality

Mortality also determines how long an individual potentially catches and consumes fish from the Site. Standard actuarial mortality tables can be used to predict the life expectancy of a given angler and whether that individual would likely remain a member of the population of living anglers. Age- and gender-specific data on mortality are available from the National Center for Health Statistics (1990) and can be used to create a complete distribution of the probability of dying at each age.

Averaging Time

Consistent with EPA (1989a) guidance, this assessment assumes a carcinogenic averaging time of 70 years. However, there is evidence to indicate that 75 years may be a more accurate estimate of lifetime (EPA, 1989c). Thus, carcinogenic exposures and risks estimated in this analysis may be overestimated in this analysis.

Point Estimate versus Probabilistic Risk Analysis

The incorporation of full distributions for exposure parameters into a Monte Carlo analysis greatly reduces the amount of uncertainty associated with risk estimates. Unlike point estimate analyses for which it is necessary to select a single descriptor for each parameter, Monte Carlo analysis allows the full range of values to be used in accordance with an assigned probability of occurrence. Thus, the multiplicative conservatism associated with point estimate analyses that use upper-bound exposure parameters is greatly reduced. Use of probabilistic techniques, such as Monte Carlo analysis, are recommended by EPA (1992) in its most recent guidelines on exposure assessment. However, a Monte Carlo analysis was beyond the scope of the current screening-level HHRA.

3.5.5 Perspective on Risk

In the risk assessment and risk management fields, health risks are defined as an estimate of the probability that a given exposure to an agent in a particular environmental setting will result in an adverse health effect (NAS, 1983; Paustenbach, 1989b). Adverse health effects may include death (mortality), illness (morbidity), or injury to individuals or a population as a whole (Graham, 1990). Historically, regulatory policy has been directed toward identifying and managing risks posed by carcinogens (EPA, 1986). A key justification for concerns over carcinogens likely stems from the fact that approximately one of every three individuals in the United States will be diagnosed with some form of cancer during their lives (i.e., a cancer incidence rate of 33%) (ACS, 1993). While noncancer effects (e.g., reproductive, immunological, etc.) are rapidly being thrust into a new category of heightened regulatory concern, carcinogens remain the highest priority.

An individual cancer risk value is an estimate of the probability that an individual member of a population will develop cancer as a result of a lifetime of exposure to a cancer-causing agent. Considering that the cumulative incidence of cancer in the U.S. population is about 33%, or 330,000 cases of cancer in 1,000,000 people (ACS, 1993), an individual exposed to a chemical

over the course of his or her lifetime resulting in an estimated incremental cancer risk level of 1 in 1,000,000 is equivalent to stating that the lifetime total cancer risk for this person is not greater than 330,001 chances in 1,000,000 (33.0001%) rather than 330,000 in 1,000,000. Clearly, the significance of 330,001 in 1,000,000 as compared to 300,000 in 1,000,000 is not in itself compelling.

Population risk, on the other hand, is a measure of the upper-limit estimate of the number of additional incidences of cancer in the exposed population (Travis et al., 1987; EPA, 1992). It is expressed as the product of the individual risk estimate and the size of the population that is potentially exposed.

Because risk management decisions involve a balancing of individual risks, population risks, and site-specific considerations (Travis et al., 1987), such decisions and remedies under the Superfund or RCRA programs of EPA are not based on a simple "bright-line" test at an individual risk level of 1×10^{-6}. In fact, these EPA programs allow for cancer risks associated with certain hazardous waste sites as high as 1×10^{-4} (EPA, 1990a). As described below, other regulatory initiatives have dealt with the "range-of-risk" approach.

3.5.5.1 Acceptable Risk Defined Under Existing Regulatory Initiatives

The foundation for risk management decisions is the selection of a cancer risk criterion which is considered to be either acceptable or *de minimis* with respect to the protection of public health and the environment. The term *de minimis* risk is used by risk assessors and regulators to define insignificant risks, or those risks that are not of regulatory concern (Travis et al., 1987). In actuality, a *de minimis* risk should be characterized as one that is judged by society to be of negligible public health concern and too small to justify the expenditure of limited risk management resources (Whipple, 1989). Often times the terms acceptable risk or *de minimus* risk are used interchangeably.

A common misconception within the field of risk assessment is that all occupational and environmental regulations adopt a theoretical maximum cancer risk of 10^{-6} as the *de minimis* or acceptable level of risk. When this criterion is exceeded, the public and the media often view the

situation as a serious public threat to public health. In 1987, Dr. Frank Young, then commissioner of the U.S. Food and Drug Administration (FDA), addressed this misconception as it related to setting tolerances for methylene chloride residues in decaffeinated coffee (Young, 1987):

> The risk level of one in one million is often misunderstood by the public and the media. It is not an actual risk; i.e., we do not expect one out of every million people to get cancer if they drink decaffeinated coffee. Rather, it is a mathematical risk based on scientific assumptions used in risk assessment. FDA uses a conservative estimate to ensure that the risk is not understated. We interpret animal test results conservatively and we are extremely careful when we extrapolate risks to humans. When FDA uses the risk level of one in one million, it is confident that the risk to humans is virtually nonexistent.

Implicit within the FDA's use of the 10^{-6} risk level for establishing a "safe level" of methylene chloride in decaffeinated coffee is the intent to protect the very large potentially exposed population of coffee drinkers. In the case of very small populations, such as pesticide applicators, *de minimis* risk levels as low as 10^{-3} for some pesticides have been deemed acceptable (Rodricks et al., 1987). In recent years, most regulatory decisions related to environmental exposure have been based on *de minimis* risk levels ranging from 10^{-4} to 10^{-6}. On the other hand, the theoretical risks associated with occupational exposure limits are usually in the range of 10^{-2} to 10^{-4} (Paustenbach, 1990a).

Acceptable Risk Under CERCLA

Final revisions to the National Contingency Plan (NCP) (EPA, 1990b) under CERCLA establish a range of 1×10^{-4} to 1×10^{-6} for generally acceptable risks at Superfund sites [40 CFR 300.430(e)(2)(i)(A)(2)]. In establishing this risk range, the EPA rejected the argument that a risk range, rather than a single risk criterion, does not adequately protect health and the environment [55 FR 8716-17, March 8, 1990]. The EPA noted that "CERCLA does not require the complete elimination of risk"; rather, remedies comply with CERCLA "when the amount of exposure is reduced so that the risk posed by contaminants is very small, i.e., at an acceptable level. EPA's risk range of 10^{-4} to 10^{-6} represents EPA's opinion on what are generally acceptable levels" [55 FR 8716]. The EPA stated that, after starting at an incremental cancer risk of 10^{-6}, selection of appropriate risks within the range should be based on "consideration of a variety of site-specific or

remedy-specific factors" [55 Fed. Reg. 8717]. According to the EPA [55 FR 8717], the appropriate factors include, but are not limited to, exposure factors, uncertainty factors, and technical factors:

> Included under exposure factors are: the cumulative effect of multiple contaminants, the potential for human exposure from other pathways at the site, population sensitivities, potential impacts on environmental receptors, and cross-media impacts of alternatives. Factors related to uncertainty may include: the reliability of alternatives, the weight of scientific evidence concerning exposures and individual and cumulative health effects, and the reliability of exposure data. Technical factors may include: detection/quantification limits for contaminants, technical limitations to remediation, the ability to monitor and control movement of contaminants, and background levels of contaminants.

Overview of Regulatory Decisions

In a retrospective review of the use of cancer risk estimates in 132 federal decisions, Travis et al. (1987) examined the cancer risks that triggered regulatory action. The authors considered three risk issues: individual risk, the size of the population exposed, and the population risk. The results of the review showed that for exposures resulting in a small-population risk, regulatory action was never taken for individual risks below 1×10^{-4}, whereas regulatory agencies almost always took action when the cancer risk exceeded approximately 4×10^{-3}. For large-population risks (e.g., the entire U.S. population), agencies typically acted on risks of about 3×10^{-4}, and *de minimis* risk was typically defined as 1×10^{-6}. These decisions demonstrate that the size of a potentially impacted population does have bearing, as it should, on the selection of acceptable risk criteria within regulatory agencies. Based on the findings of Travis et al. (1987), and upon further examination of the database, Graham (1990) has suggested using a range of 1×10^{-4} to 1×10^{-6} for acceptable lifetime cancer risk for the average exposed individual and a less stringent risk range for smaller, more highly exposed sub-populations of the general population.

As indicated in the above discussion, cancer risk levels deemed acceptable have been a function of a number of factors, including the size and characteristics of the potentially affected population, and other factors such as technical feasibility. Therefore, single cancer risk values do not provide

flexibility for making risk management decisions on a case-by-case basis. An acceptable risk range is more appropriate for determining site-specific remedies.

In comparison to background incidences of cancer in the U.S. population, incremental risks of 10^{-4} to 10^{-6} are negligible. The background incidence of all cancers in the U.S. population is approximately 33%, or 3.3 in 10 (ACS, 1992). Thus, an incremental risk level of 1×10^{-4} would indicate that a given lifetime exposure would increase the potential lifetime cancer risk from approximately 33% to 33.01%.

3.5.5.2 Comparative Costs

While risk assessment provides a quantitative estimate of the potential health threat associated with a given situation, risk management strives to balance the social, political, and economic facets of a given situation (CEQ, 1989). In selecting an acceptable risk level for setting site remediation goals, economic factors (i.e., the cost of remediation) become the most important of these additional considerations.

When choosing appropriate risk levels, regulators should weigh the economic costs and benefits that may be associated with risk reduction. Although some environmental laws attempt to restrict economic considerations, common sense and studies of regulatory behavior indicate that economic factors play a critical role in environmental decision making. The economic consequences of regulatory decisions must be heeded so that public health is not adversely affected. Public health professionals have recognized for decades that reducing family income impairs public health (Graham, 1990). The costs of environmental regulation may reduce real family income by increasing the prices of goods and services that all of us purchase, which ultimately causes a reduction in real family incomes. Subsequently, when families have less income, they have less money available for everything from preventive checkups to smoke detectors. If regulatory costs are excessive, the regulator may inadvertently cause more harm to the health status of families than will be prevented.

The justification for integrating comparative remedial cost estimates into the establishment of a site-specific acceptable risk level is tied to the concept that, in most cases, there is little difference between a 10^{-6} risk level and a 10^{-5} risk level or between a 10^{-5} risk level and a 10^{-4} risk level, in

terms of real human health risks. In fact, although there may be little difference in real health risk, there may be a significant difference in remedial costs associated with one risk level as compared to another.

3.6 Summary and Conclusions

In accordance with the IWP and consistent with EPA guidance (1987, 1989a,b,c, 1991a, 1992, 1993b,c, 1994a), a screening-level human health risk assessment was conducted to evaluate the potential health risks associated with human exposures to chemicals in sediment and water in the Passaic River Study Area. Using available analytical data for surface sediments, 88 CPC were selected for assessment based on methods recommended by EPA (1989a), including an evaluation of essential nutrients, and results of concentration-toxicity and bioaccumulation screens. Potential exposure via surface water, edible tissues of biota, and sediments were considered in the exposure assessment. Evaluation of Site conditions, past and present land use, and demographic information, indicate that human populations potentially exposed to chemicals at the Site are limited to urban recreational anglers who may consume fish from the Site. For estimation of exposure to CPC from consumption of fish, exposure point concentrations in edible tissue of indicator species (striped bass and blue crab) were estimated from the 95% UCL of the arithmetic mean of the Site sediment data using a food web model and empirical relationships. Intakes (mg/kg-day) for typical and reasonable maximum exposure (RME) for CPC were estimated using exposure models and parameters recommended in EPA (1989a,b,c) guidance, and available data from the peer-reviewed scientific literature regarding consumption of fish by urban populations. Consistent with EPA (1989a) guidance, carcinogenic risk and noncarcinogenic hazard were characterized by combining intakes for CPC with chemical-specific toxicity criteria (i.e., cancer slope factors for carcinogens and reference doses for noncarcinogens) obtained from EPA (1994a, 1995) and ECAO (1995).

Based on the results of this screening-level assessment, cumulative hypothetical upper-bound incremental carcinogenic risks associated with consumption of striped bass and blue crab from the Site are estimated to be 7×10^{-6} for typical exposure and 3×10^{-4} for reasonable maximum exposure (RME). Chemicals contributing the majority of cumulative risk include: arsenic (62%); beryllium (12%); 2,3,7,8-TCDD (9.0%); and 3,3' 4,4'-TetraCB (4.3%). Total noncarcinogenic hazards from consumption of striped bass and blue crab from the Site were estimated to be 0.27 for typical exposure and 3.3 for the RME. Based on this analysis, the majority of noncancer

hazards are associated with: arsenic (24%); monobutyltin (12%); antimony (12%); Aroclors 1248 (8.8%); aluminum (7.0%); mercury (6.2%); dibutyltin (5.4%); thallium (4.3%); cadmium (3.5%); copper (3.4%); vanadium (2.9%); Aroclor 1254 (2.1%); barium (1.6%); nickel (1.6%); manganese (1.5%); and zinc (1.0%). Based on the results of the uncertainty analysis that was performed as part of the risk characterization, ChemRisk concludes that the screening-level risk estimates presented in this evaluation are almost certain to overstate the actual risks by a considerable margin.

3.7 Recommendations

Following review by Environmental Protection Agency personnel of the detailed basis for, and inherent uncertainties in, the predicted human health risks presented in this report, a meeting among respondent and agency personnel, in a technical workshop format, would be appropriate to assess the useability of the HERA process and results of this study, including means to reduce the uncertainty in the screening-level assessment. A workshop will serve to focus comments, and should accelerate a mutual understanding on how to complete a final HERA.

3.8 References for Section 3.0

ACS. 1992. *Cancer Facts and Figures - 1992.* American Cancer Society, Atlanta, GA.

ACS. 1993. *Cancer Facts & Figures - 1993.* American Cancer Society, Atlanta, GA.

ATSDR. 1991. *Toxicological Profile for Cyanide; Draft for Public Comment.* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. October.

Barnes, D.G. and M. Dourson. 1988. Reference dose (RfD): Description and use in health risk assessments. *Regul. Toxicol. Pharmacol.* 8:471-486.

Belton, T.J., B. Ruppel, K. Lockwood, and M. Boriek. 1983. *PCBs in Selected Finfish Caught Within New Jersey Waters 1981-1982 (With Limited Chlordane Data).* New Jersey Department of Environmental Protection, Trenton, NJ. July 1.

Belton, T. J., R. Hazen, B.E. Ruppel, K. Lockwood, R. Mueller, E. Stevenson, and J.J. Post. 1985. *A Study of Dioxin (2,3,7,8-Tetrachlorodibenzo-p-Dioxin) Contamination in Select Finfish, Crustaceans, and Sediments of New Jersey Waterways.* New Jersey Department of Environmental Protection, Office of Science and Research, Trenton, NJ. October 30.

Boyle, K.J., R.K. Roper, and S.D. Reiling. 1990. *Highlights From the 1988 Survey of Open Water Fishing in Maine.* University of Maine, Department of Agricultural and Resource Economics, Orono, ME. July.

Brydon, N.F. 1974. *The Passaic River: Past, Present, Future.* New Brunswick, New Jersey: Rutgers University Press.

CEQ. 1989. *Risk Analysis: A Guide to Principles and Methods for Analyzing Health and Environmental Risks.* U.S. Council on Environmental Quality.

ChemRisk. 1992. *Consumption of Freshwater Fish by Maine Anglers.* ChemRisk® - A Division of McLaren/Hart, Portland, ME. July 24.

ChemRisk. 1994. *Estimating Fish Consumption Rates for the Upper Hudson River.* ChemRisk® - A Division of McLaren/Hart, Portland, ME.

Connelly, N.A., B.A. Knuth, and C.A. Bisogni. 1992. *Effects of the Health Advisory Changes on Fishing Habits and Fish Consumption in New York Sport Fisheries.* Human Dimension Research Unit, Department of Natural Resources, New York State College of Agriculture and Life Sciences, Fernow Hall, Cornell University, Ithaca, NY. Report for the New York Sea Grant Institute Project No. R/FHD-2-PD. September.

Covello, V.T. 1989. Communicating right-to-know information on chemical risks. *Environ. Sci. Technol.* 23(12):1444-1449.

Crawford, D.W., N.L. Bonnevie, C.A. Gillis, and R.J. Wenning. 1994. Historical changes in the ecological health of the Newark Bay Estuary, New Jersey. *Ecotoxicol. Environ. Saf.* 29:276-303.

Crump, K., B. Allen, and A. Shipp. 1989. Choice of dose measure for extrapolating carcinogenic risk from animals to humans: An empirical investigation of 23 chemicals. *Health Phys.* 57(1):387-393.

Dourson, M.L. and J.F. Stara. 1983. Regulatory history and experimental support of uncertainty (safety) factors. *Regul. Toxicol. Pharmacol.* 3:224-238.

Dourson, M.L., L.A. Knauf, and J.C. Swartout. 1992. On reference dose (RfD) and its underlying toxicity data base. *Toxicol. Ind. Health* 8(3):171-189.

Ebert, E.S., N.W. Harrington, K.J. Boyle, J.W. Knight, and R.E. Keenan. 1993. Estimating consumption of freshwater fish among Maine anglers. *N. Am. J. Fish. Manage.* 13(4):737-745.

Ebert, E.S., P.S. Price, and R.E. Keenan. 1994. Selection of fish consumption estimates for use in the regulatory process. *J. Exp. Anal. Environ. Epid.* 4(3):373-393.

ECAO. 1995. *Correspondence with Marion Olsen.* U.S Environmental Protection Agency's Environmental Criteria and Assessment Office, Cincinnati, Ohio.

EPA. 1986. *Guidelines for Carcinogen Risk Assessment.* U.S Environmental Protection Agency, Washington, D.C., 51 Federal Register 185:33992-34003. September 24.

EPA. 1987. *The Risk Assessment Guidelines of 1986.* U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C. EPA/600/8-87/045. August.

EPA. 1989a. *Risk Assessment Guidance for Superfund; Volume I: Human Health Evaluation Manual (Part A) - Interim Final.* U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-89-002. July.

EPA. 1989b. *Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish: A Guidance Manual.* U.S. Environmental Protection Agency, Office of Marine and Estuarine Protection, Washington, D.C. EPA-503/8-89-002. September.

EPA. 1989c. *Exposure Factors Handbook.* U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C. EPA/600/8-89/043. July.

EPA. 1989d. *Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-*p*-dioxins and -Dibenzofurans (CDDs and CDFs) and 1989 Update.* U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, D.C. EPA/625/3-89/016. March.

EPA. 1990a. *National Contingency Plan.* U.S. Environmental Protection Agency, Washington, D.C.

EPA. 1990b. *The Report of the Human Health Subcommittee: Relative Risk Reduction Project - Reducing Risk, Appendix B.* U.S. Environmental Protection Agency, Science Advisory Board, Washington, D.C. PB91-155267. September.

EPA. 1991a. *Assessment and Control of Bioconcentratable Contaminants in Surface Waters.* U.S. Environmental Protection Agency, Office of Water, Washington, D.C. March.

EPA. 1991b. *Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual Supplemental Guidance "Standard Default Exposure Factors".* U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Toxics Integration Branch,Washington, D.C. EPA 540/1-89/002. March 25.

EPA. 1992. *Final Guidelines for Exposure Assessment; Notice.* U.S. Environmental Protection Agency, Washington, D.C., 57 Federal Register 104:22888-22938. May 29.

EPA. 1993a. *Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife.* U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C. EPA/600/R-93/055. March.

EPA. 1993b. Memo to the Directors of Waste Management Division, Emergency and Remedial Response Division, Hazardous Waste Management Division, and Hazardous Waste Division from W.H. Farland and H.L. Longest, U.S. Environmental Protection Agency, Washington, D.C. Re: Use of IRIS Values in Superfund Risk Assessment. OSWER Directive #9285.7-16. December 21.

EPA. 1993c. *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons.* U.S. Environmental Protection Agency, Research and Development, Washington, D.C. ECAO-CN-842. March.

EPA. 1994a. *Health Effects Assessment Summary Tables FY-1994 Annual.* U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, D.C. PB94-921199. March.

EPA. 1994b. *Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds, Volumes 1 through 3.* U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C. EPA/600/BP-92/001a. June.

EPA. 1995. *Integrated Risk Information Service.* U.S. Environmental Protection Agency, Cincinnati, OH.

Graham, J.D. 1990. Letter to Dean C. Marriott. Commissioner, Department of Environmental Protection for the State of Maine, Augusta, ME. October.

Hall, W.S. and G.W. Pulliam. 1995. An assessment of metals in an estuarine wetlands ecosystem. *Arch. Environ. Contam. Toxicol.* (submitted)

Hauge, P., J. Bukowski, P. Morton, M. Boriek, and G. Casey. 1990. *Polychlorinated Biphenyls (PCBs), Chlordane, and DDTs in Selected Fish and Shellfish from New Jersey Waters, 1986-1987: Results from New Jersey's Toxics in Biota Monitoring Program.* New Jersey Department of Environmental Protection, Trenton, NJ.

Hauge, P., J. Bukowski, P. Morton, M. Boriek, and G. Casey. 1993. *Polychlorinated Biphenyls (PCBs), Chlordane, and DDTs in Selected Fish and Shellfish from New Jersey Waters, 1988-1991: Results from New Jersey's Toxics in Biota Monitoring Program.* New Jersey Department of Environmental Protection, Trenton, NJ.

Javitz, H. 1980. *Seafood Consumption Data Analysis; Final Report.* Prepared by Statistical Analysis Department, SRI International, Menlo Park, CA for H.D. Kahn, U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, D.C. Task 11, EPA Contract No. 68-01-3887. September 24.

Keenan, R.E., B.L. Finley, and P.S. Price. 1994. Exposure Assessment: Then, now, and quantum leaps in the future. *Risk Analysis* 14(3):225-230.

Klaassen, C.D., M.O. Amdur, and J. Doull. 1986. *Casarett and Doull's Toxicology: The Basic Science of Poisons.* New York: Macmillan Publishing Company.

Landolt, M.L., F.R. Hafer, A. Nevissi, G. van Belle, K. Van Ness, and C. Rockwell. 1985. *Potential Toxicant Exposure Among Consumers of Recreationally Caught Fish from Urban Embayments of Puget Sound: Final Report.* National Oceanic and Atmospheric Administration, Rockville, MD. NOAA Tech. Memo. NOS OMA 33. November.

Landolt, M., D. Kalman, A. Nevissi, G. van Belle, K. Van Ness, and F. Hafer. 1987. *Potential Toxicant Exposure Among Consumers of Recreationally Caught Fish from Urban Embayments of Puget Sound; Final Report.* National Oceanic and Atmospheric Administration, Rockville, MD. NOAA Technical Memorandum NOS OMA 33. April.

Lewis, S.C., J.R. Lynch, and A.I. Nikiforov. 1990. A new approach to deriving community exposure guidelines from no-observed-adverse-effect levels. *Reg. Toxicol. Pharmacol.* 11:314-330. (cited in Dourson et al., 1992).

Lunde, G. 1977. Occurrence and transformation of arsenic in the marine environment. *Environ. Health Persp.* 19:47-52.

McCormick, M. and P.T. Quinn. 1975. Phytoplankton diversity and chlorophyll-a in a polluted estuary in Newark Bay, NJ. *Mar. Poll. Bull.* 6:105-108.

Munro, I.C. and D.R. Krewski. 1981. Risk assessment and regulatory decision making. *Fd. Cosmet. Toxicol.* 19:549-560.

Nagao, T., G. Golor, H. Hagenmaier, and D. Neuseri. 1993. Teratogenic potency of 2,3,7,8-pentachlorodibenzofuran and of three mixtures of polychlorinated dibenzo-p-dioxins and dibenzofurans in mice. *Arch. Toxicol.* 67:591-597.

National Center for Health Statistics. 1990. *Vital and Health Statistics.* Hyattsville, MD.

NAS. 1983. *Risk Assessment in the Federal Government: Managing the Process.* Washington, D.C.: National Academy Press.

Neubert, R., G. Golor, R. Stahlmann, H. Helge, and D. Neubert. 1992. Polyhalogenated dibenzo-p-dioxins and dibenzofurans and the immune system: 4. Effects of multiple-dose treatment with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on peripheral lymphocyte subpopulations of a non-human primate (*Callithrix jacchus*). *Arch. Toxicol.* 66:250-259.

NJDEP. 1992. *State Water Quality Inventory Report*. New Jersey Department of Environmental Protection, Division of Water Resources, Trenton, NJ. September.

NJDEP. 1995. Community-based educational outreach to at-risk urban anglers. In: *Second Annual Marine and Estuarine Shallow Water Science and Management Conference.* Holiday Inn on the Boardwalk, Atlantic City, NJ. April 3-7.

NJMSC. 1987. *The Hudson-Raritan: State of the Estuary. Summary Vol. 1, Part 1.* Reported by The Panel on Water Quality of the Hudson-Raritan Estuary. New Jersey Marine Sciences Consortium, Trenton, NJ. December.

NOAA. 1981. *Water Quality of the Hudson-Raritan Estuary-Draft.* National Oceanic and Atmospheric Administration, Office of Marine Pollution Assessment, Boulder, CO. NOAA Grant # NA8ORAD00034. November.

NOAA. 1990. *Contaminants in Sediment and Fish Tissue from Estuarine and Coastal Sites of the Northeastern United States: Data Summary for the Baseline Phase of the National Status and Trends Program Benthic Surveillance Project, 1984 - 1986.* National Oceanic and Atmospheric Administration, Rockville, MD. Technical Memorandum NMFS-F/NEC-79. December.

NOAA. 1995. *Chemical Contaminant Levels in Flesh of Four Species of Recreational Fish from the New York Bight Apex.* National Oceanic and Atmospheric Administration, National Marine Fisheries Service, Highlands, NJ. January 27.

NYSDEC. 1991. *New York State 1993-94 Fishing Regulations Guide.* New York State Department of Environmental Conservation, Albany, NY.

O'Connor, J.M. and J.M. Rachlin. 1982. Perspectives on metals in New York Bight organisms: Factors controlling accumulation and body burdens. In: *Ecological Stress and the New York Bight: Science and Management.* G.F. Mayer (eds.). Rockville, MD: National Oceanic and Atmospheric Administration. 655-674.

Paustenbach, D.J. 1989a. *The Risk Assessment of Environmental and Human Health Hazards: A Textbook of Case Studies.* New York, NY: John Wiley and Sons.

Paustenbach, D.J. 1989b. Important recent advances in the practice of health risk assessment: Implications for the 1990's. *Regul. Toxicol. Pharmacol.* 10:204-243.

Paustenbach, D.J. 1990a. Health risk assessment and the practice of industrial hygiene. *Am. Ind. Hyg. Assoc. J.* 51(7):339-351.

Paustenbach, D.J. 1990b. Occupational exposure limits: Their critical role in preventive medicine and risk management. *Am. Ind. Hyg. Assoc. J.* 51(May).

Pearce, N., A.H. Smith, and J.S. Reif. 1988. Increased risks of soft tissue sarcoma, malignant lymphoma, and acute myeloid leukemia in abattoir workers. *Am. J. Ind. Med.* 14:63-72.

Pierce, D., D. Noviello, and S.H. Rogers. 1981. *Commencement Bay Seafood Consumption Study; Preliminary Report.* Tacoma-Pierce County Health Department, Seattle, WA. December.

Price, P.S., S.H. Su, and M.N. Gray. 1994. The effect of sampling bias on estimates of angler consumption rates in creel surveys. *J. Exp. Anal. Environ. Epid.* 4(3):355-372.

Puffer, H.W., S.P. Azen, M.J. Duda, and D.R. Young. 1981. *Consumption Rates of Potentially Hazardous Marine Fish Caught in the Metropolitan Los Angeles Area.* Prepared by the University of Southern California School of Medicine for the U.S. Environmental Protection Agency, Office of Research and Development, Environmental Research Laboratory, Corvallis, OR. Grant No. R 807 120010.

Reinert, R.E., D. Stewart, and H.L. Seagran. 1972. Effects of dressing and cooking on DDT concentrations in certain fish from Lake Michigan. *J. Fish. Res. Board Can.* 29:525-529.

Rodricks, J.V., S.M. Brett, and G.C. Wrenn. 1987. Significant risk decisions in federal regulatory agencies. *Regul. Toxicol. Pharmacol.* 7:307-320.

Rupp, E.M. 1980. Age dependent values of dietary intake for assessing human exposures to environmental pollutants. *Health Physics* 39:151-163.

Safe, S., D. Davis, M. Romkes, C.Yao, B. Keyes, J. Piskorska-Pliszczynska, K. Farrell, G. Mason, M.A. Denomme, L. Safe, B. Zmudzka, and M. Holcomb. 1989. Development and validation of in vitro bioassays for 2,3,7,8-TCDD equivalents. *Chemosphere* 19(1-6):853-860.

Safe, S. 1990. Polychlorinated biphenyls (PCBs), dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), and related compounds: Environmental and mechanistic considerations which support the development of toxic equivalency factors (TEFs). *CRC Crit. Rev. Toxicol.* 21(1):51-88.

Sherer, R.A. and P.S. Price. 1993. The effect of cooking processes on PCB levels in edible fish tissue. *Qual. Assur. Good Pract. Reg. Law* 2(4):396-407.

Smith, M.H., J.B. Gentry, and J. Pinder. 1973. Annual fluctuations in small mammal populations in an eastern hardwood forest. *J. Mammal.* 55(1):231-234.

Stachiw, N.C., M.E. Zabik, A.M. Booren, and M.J. Zabik. 1988. Tetrachlorodibenzo-p- dioxin residue reduction through cooking/processing of restructured carp fillets. *J. Agric. Fd. Chem.* 36(4):848-852.

Travis, C.C., S.A. Richter, E.A.C. Crouch, R. Wilson, and E.D. Klema. 1987. Cancer risk management: A review of 132 federal regulatory decisions. *Environ. Sci. Technol.* 21(5):415-420.

U.S. Bureau of the Census. 1988. *Geographical Mobility: March 1985 to March 1987.* U.S. Department of Commerce, Economics and Statistics Administration, Bureau of the Census, Washington, D.C. Series P-20. December.

U.S. Bureau of the Census. 1991. *Geographical Mobility: March 1987 to March 1990.* U.S. Department of Commerce, Economics and Statistics Administration, Bureau of the Census, Washington, D.C. Series P-20, No. 456. December.

Whipple, C. 1989. Nonpessimistic risk assessment and de minimis risk as risk management tools. In: *The Risk Assessment of Environmental and Human Health Hazards: A Textbook of Case Studies.* D.J. Paustenbach (ed.). New York, NY: John Wiley & Sons.

Wilson, R. 1979. Analyzing the daily risks of life. *Tech. Rev.* 81(4):41-46.

Young, F.E. 1987. Risk assessment: The convergence of science and the law. *Regul. Toxicol. Pharmacol.* 7:179-184.

Zabik, M.E., C. Merrill, and M.J. Zabik. 1982. Predictability of PCBs in carp harvested in Saginaw Bay, Lake Huron. *Bull. Environ. Contam. Toxicol.* 28:592-598.

Zabik, M.E. and M.J. Zabik. 1995. Tetrachlorodibenzo-p-dioxin residue reduction by cooking/processing of fish fillets harvested from the Great Lakes. *Bull. Environ. Contam. Toxicol.* 55:264-269.

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4.0 SCREENING-LEVEL ECOLOGICAL RISK ASSESSMENT

Ecological risk assessment is a process that evaluates the likelihood that adverse ecological effects may occur or are occurring as a result of exposure to one or more stressors (EPA, 1992a). While ecological risk assessments cannot provide absolute proof of the occurrence of adverse impacts or the lack thereof (EPA, 1989), they can provide the quantitative basis for comparing and prioritizing risks, as well as a systematic means of improving the understanding of risks (Suter, 1993). They can be used to help identify environmental problems, establish priorities, and provide a scientific basis for selecting remedial options (EPA, 1992a).

In Section 4.0, a baseline screening-level ecological risk assessment (ERA) is presented that evaluates the potential impacts of CPC and other stressors on key organisms at the Site. The primary objectives of the ERA were twofold: 1) to utilize the available sediment and water quality data, other available literature, and unpublished environmental data to characterize potential impacts to key organisms posed by physical and chemical stressors at the Site; and 2) to evaluate the available site-specific literature regarding current or past environmental conditions in the river, particularly those which may affect the exposure of key organisms to chemical and water quality stressors.

The EPA (1992a) recommends conducting assessments of ecological risks in three stages, consisting of Problem Formulation, Analysis, and Risk Characterization. During Problem Formulation (Section 4.1), the goals and focus of the ecological risk assessment are established, Site-specific factors that define the feasibility, scope and objectives of the ecological risk assessment are presented (EPA 1991, 1992a). Problem formulation for the Site has been addressed in the AOC and SOW, and is summarized in Section 4.1, as are the Site-specific factors that define the feasibility, scope, and objectives of the screening-level ERA.

Consistent with the second element of EPA's framework (1992a), Analysis, a technical evaluation of data on the potential effects of and exposure to the environmental stressors (in this case, chemicals in sediments and water and physical stressors) was performed. The analysis consists of an ecological community characterization (Section 4.2), selection of chemicals of potential concern (Section 4.3), exposure assessment (Section 4.4), and an ecological effects assessment (Section 4.5).

Consistent with the third element of EPA's framework (1992a), Risk Characterization, the results of the analysis were used to assess the likelihood of adverse impacts associated with the exposure of key organisms to environmental stressors (EPA, 1991, 1992a). The risk characterization (Section 4.5) also includes a summary of assumptions used, scientific uncertainties, and strengths and weaknesses of the analysis (EPA, 1989, 1991, 1992a).

4.1 Problem Formulation

Problem Formulation for the Site was conceptually addressed in the AOC and SOW. Consistent with the IWP, the AOC and SOW were used as the conceptual model for implementing the screening-level ecological risk assessment for the Site. The historical physical and chemical stressors that have resulted in the degradation of the ecology of the Site over the past two centuries are discussed in this Section. These stressors, which may contribute substantially to the potential ecological risks at the Site, include chemical contamination of sediments and surface water, and physical alterations of the shallow water habitats that were once present at the Site. The potential risks associated with chemical stressors that are currently present in surface sediments, water, and aquatic organisms at the Site are evaluated in the Analysis and Risk Characterization.

4.1.1 Historical Contamination of Sediments and Surface Water

The quality of marine and estuarine resources in many coastal regions, particularly those in metropolitan areas, is frequently threatened by sediment and water contamination associated with high density urban development (Mytelka et al., 1973; Meyerson et al., 1981). The high levels of toxic chemicals that have been reported in the seawater and bottom sediments of numerous harbors have been associated with wastes from a wide variety of urban, industrial, and riverine sources (NOAA, 1991). The metropolitan region surrounding the Site has been recognized as the largest manufacturing and industrial center in the eastern United States since the early 1800s. For many years, a myriad of activities have resulted in shoreline development/modifications and pollution from municipal and industrial wastewaters, stormwater runoff, accidental spills, direct dumping of wastes, and atmospheric deposition (Olsen et al., 1984; HydroQual, 1991). These changes have had a substantial impact on the ecological conditions of the Site. A detailed discussion of these impacts is presented in Crawford et al. (1994, 1995), and a summary is provided below.

4.1.1.1 Water Quality

Degradation of water quality in the Passaic River, including the Site, first became apparent during the Civil War (Brydon, 1974). By 1872, official reports described water "highly offensive to both smell and taste", and having "a shocking degree of contamination", and a "filthy appearance" (Cunningham, 1966a,b). According to Galishoff (1988), complaints "were received from all sections of the city that the water smelled like creosote, tasted bad, and had a bad odor." In 1873, coal tar residues suspended in the river water were noted (Brydon, 1974). The deteriorating water quality of the Passaic River during this period forced many residents to dig their own wells; by 1885 however, a survey showed that seventy-five percent of groundwater wells also were polluted (Cunningham, 1966a,b). Between 1884 and 1890, over 1,000 of Newark's more than 1,500 wells had been closed due to contamination (Galishoff, 1988). In 1887, an inspector for the Passaic River declared that legal action would be required to mitigate pollution of the river from industrial waste practices (Brydon, 1974).

The population expansion during the nineteenth century and first half of the twentieth century resulted in the generation of increasing volumes of human wastes. The Passaic River was often characterized as an open sewer (Suszkowski et al., 1990). In 1894, as much as one third of the total flow of the Passaic River was estimated to be sewage (Brydon, 1974). In 1910, the mouth of the Passaic River was declared to be "black from the sewage and manufacturing wastes it receives" (Mytelka et al., 1981). Efforts to improve the water quality and to reduce the spread of disease of the Passaic River led to the construction of a trunk sewer line system in 1924 (Brydon, 1974). However, despite the development of sewage treatment plants, many industrial facilities located along the Passaic River were not connected to the trunk line until the late 1950s (Brydon, 1974).

Excessive loadings of conventional pollutants such as total suspended solids, organic matter, nitrogen, ammonia, and pathogens associated with wastewater discharges and their impact on dissolved oxygen (DO) levels in the River has been a chronic problem at the Site since the early 1900s (API, 1972; McCormick et al., 1983). Investigations conducted prior to 1940 by the Interstate Sanitation Commission (ISC) indicated substantially decreased levels of DO throughout the region during the early part of the century (ISC, 1939). For example, DO measurements

collected in 1909 showed that the Passaic River did not meet minimum fish survival standards (0.00 to 2.89 mg/l, with an average of 0.33 mg/l) (Mytelka et al., 1981). Recently, low DO levels were still reported to occur at the Site (ChemRisk, 1995a).

4.1.1.2 Sediment Quality

The problem of chemicals in sediments at the Site has been the focus of much concern in recent years (Meyerson et al., 1981; IT, 1986; Finley et al., 1990; Bonnevie et al., 1992; Gillis et al., 1993; Huntley et al., 1993; Bonnevie et al., 1994; Wenning and Erickson, 1994; Gillis et al., 1995; Iannuzzi et al., 1995; Huntley et al., 1995). During the 1980s and early 1990s, several investigations were conducted to evaluate the concentrations of various chemicals in sediments at the Site. These include investigations conducted as part of the Diamond Alkali Superfund Site investigation, investigations conducted on behalf of Occidental Chemical Corporation in the early 1990s, and investigations conducted by various governmental agencies including the ACOE and EPA. These investigations revealed that sediments from the Site contain elevated concentrations of a myriad of hazardous chemicals including, but not limited to, heavy metals, pesticides, PCBs, PCDD/Fs, PAHs, petroleum hydrocarbons, and other volatile and semi-volatile organic compounds. The historical and current mass loading of such chemicals is associated with several sources including, but not limited to, POTWs and CSOs, industrial waste discharged either directly to the estuary or through POTWs, stormwater runoff, and accidental spills of petroleum products and hazardous chemicals.

4.1.2 Historical Alterations of the Site Ecosystem

During the past two centuries, the Site has been subject to multiple influences and changes due not only to natural physical (hydrological, topographical, and climatological) and ecosystem progressions, but also to the pressures exerted by rapidly expanding urban and industrial development in the region. Within the last two decades, the ecological conditions of this region have been the focus of an increasing number of studies (NJMSC, 1987; NOAA, 1988; Squires and Barclay, 1990; ChemRisk 1995a,b). Some of these studies examined ecological impacts within the entire NY/NJ Harbor Estuary, of which the Site is a small portion. Those studies encompassing the Site have indicated that adverse impacts on the ecological health of the estuary, particularly reduced diversity and abundance of organisms, are the result of historical urban-industrial activities

(NJMSC, 1987; Pearce, 1988). The State of New Jersey has issued several advisories on eating sport fish and wildlife taken from the Site environs because some contain elevated levels of chemicals, including PCBs, mercury, chlordane, and PCDD/Fs (Kennish et al., 1992).

Despite initial indications of declining water quality, the tidal Passaic River was considered a prime fishing area in New Jersey in the early and mid-1880s (Brydon, 1974). An extensive shad fishery existed in the River in the early and mid-1800s (Brydon, 1974). In addition to shad, species such as herring, chub, suckers, bass, pickerel, sturgeon, sunfish, white and yellow perch, mussels, and eels were commonly found in the River (Brydon, 1974).

A significant commercial fishery has not operated within the tidal Passaic River since the early 1900s (McCormick and Quinn, 1975). Originally, the decline in fishing was associated with an increasing awareness of pollution; as early as the Civil War, sales of oysters and shad from the region were affected by reports that the organisms were tainted with coal oil (Earll, 1887). According to one author, the shad catch decreased by 84% from 1880 to 1908, largely as a result of "off flavors" (Squires, 1981). Populations of commercial species were also substantially reduced from both overharvesting (Mytelka et al., 1981; Esser, 1982; Franz, 1982) and pollution (Esser, 1982). As early as 1885, the Commission of Fisheries of New Jersey reported that water-borne pollution was resulting in declining populations of shad in the tidal Passaic River (Esser, 1982). After the turn of the century, conditions apparently deteriorated rapidly until 1926, when a survey conducted in the area by the US War Department found "fish life destroyed" (Hurley, 1992).

Few data regarding populations of fish exist for the remainder of the twentieth century. In general, it appears that populations have remained in decline; however, some species, such as striped bass, have recently been collected in the River (NJDEP, 1993). A characterization of the current ecological community at the Site is presented in Section 4.2.

Industrial and urban activity surrounding the Site has resulted in a severe reduction in the availability of natural habitats for indigenous and migratory biota. Nearly all of the original tidal marsh and wetland areas that were once present at the Site have been filled or dredged, while the majority of those remaining have been significantly altered by a variety of human activities (Squires and Barclay, 1990). In addition, much effort has been directed towards stabilizing river banks,

and redirecting water flow through the construction of dikes or dams, such that alterations in erosion and sedimentation patterns have occurred (Squires, 1992). For example, the Dundee Dam was built in 1859 on the Passaic River to generate electrical power (Brydon, 1974). Dredging activities and channel improvements, which began in 1874 (ACOE, 1988), have also continued the alteration of ecological conditions at the Site (Wallace and Wallace, 1983; Burger et al., 1993). The shipping channels in the lower Passaic River were frequently dredged after 1900, most recently in 1989.

Much of the city of Newark, NJ occupies land once dominated by salt marsh, which was filled with more than 21 million tons of material, including industrial and municipal wastes, dredge spoils, and railroad cinders (Zdepski, 1992). The southern bank of the lower Passaic River, just upstream of the NJ Turnpike Bridge was once primarily marshland. ERM (1990) reports that between 1873 and 1890, this area was extensively filled with 8 to 12 feet of mixed fill material from coal-gasification facilities, eliminating the marsh habitat, and introducing various organic contaminants such as PAHs (ERM, 1992). By the early 1900s, numerous other salt marshes were filled with solid waste in an effort to eliminate mosquito breeding areas (Zdepski, 1992). Increasing urbanization has also resulted in the application of pesticides that are toxic to aquatic organisms for the control of urban and suburban pests (Rod et al., 1989). The loss of habitat typically results in deteriorating conditions for populations of aquatic organisms (Purves and Orians, 1983) and can have far-reaching implications on the entire ecosystem, as the structure and function of aquatic communities are affected.

The available historical information, while limited in many respects, records that historical shoreline development/modifications and pollutant loadings throughout this century have had a substantial adverse impact on the ecological conditions of the Site environs (McCormick and Quinn, 1975; Earll, 1887; Mytelka et al., 1981; Esser, 1982; Squires, 1981; Hurley, 1992). The current status of the Site ecology should be properly viewed as a product of long term, accumulative, adverse impacts as the result of more than 100 years of multiple influences and changes, many of them irreversible.

4.1.3 Stressor Characterization

An estuary is an enclosed or partly enclosed coastal body of water that is connected with the open sea and within which sea water is diluted with freshwater drainage from the estuary watershed. The salinity and density gradients created by mixtures of seawater and freshwater in an estuary, as well as the harsh and dynamic environmental conditions produced by semi-diurnal tides, are responsible for the unique ecological attributes of estuaries that place significant physiological demands on biota. Estuaries are naturally characterized by large populations of relatively few species due to the relative small number of species that are tolerant of such dynamic environmental conditions (Levinton, 1982). A healthy estuary normally supports large, fluctuating populations of phytoplankton, invertebrates, fishes, and fish-eating wildlife such as waterfowl and semi-aquatic mammals. The Site, by contrast, is not a healthy estuarine habitat, primarily due to the presence of major physical and chemical stressors, as summarized below.

4.1.3.1 Historical Physical Alterations to the Site Ecosystem

The geographical alterations to the tidal Passaic River since the 1800s are responsible for much of the destruction of the habitat necessary for the maintenance of aquatic and avian populations (Wallace and Wallace, 1983; Burger et al., 1993). Substantial reductions of different habitats within the estuary (e.g., salt marsh, soft bottom substrate, and rocky intertidal) have eliminated or reduced the resources that are necessary to sustain many species, and have created more competition among the remaining species. For instance, estuaries are particularly important as nurseries for juveniles of many fish and invertebrate species; in fact, on the Atlantic Coast of the U.S., the majority of commercially exploited fish species utilize estuaries as juvenile feeding grounds (Levinton, 1982). However, the nursery function of an estuary is directly related to the amount of salt marsh habitat that is associated with a particular waterway (Weinstein et al., 1980; Boesch and Turner, 1984). The removal of nearly all of the salt marsh that was historically associated with the Site has eliminated the ability of the Site to provide nursery habitat or sufficient cover for migratory fishes and crustaceans. In addition, the habitat and food supply for waterfowl and semi-aquatic mammals has been reduced to negligible levels. Because these changes are irreversible, given the current land use surrounding the Site, loss of habitat is currently the primary stressor to the Site ecosystem.

4.1.3.2 Dissolved Oxygen

Depressed levels of DO, resulting primarily from chemical and biological oxygen demands created by discharges of wastes (containing nutrients and hazardous chemicals) to the Site, adversely affect fish and benthic invertebrate communities by inhibiting growth, decreasing survival rates, and increasing competition (Stacey, 1990). In general, low DO concentrations have been found to result in reduced species abundance and diversity in estuarine environments (Boesch and Rosenburg, 1981; Keller and Squibb, 1992). Episodic fish kills observed during the past century have often been attributed to hypoxia (Sindermann and Swanson, 1979; Padar, 1990).

It has been reported that dissolved oxygen levels between 0 - 0.5 mg/l are lethal to most species of fish and benthos (Theede et al., 1969; McCarthy, 1969; Saksena and Joseph, 1972; Shumway et al., 1983; Stickle et al., 1989). Many species of crustaceans are extremely sensitive to hypoxic conditions and may show increased mortality in waters with DO concentrations which are only slightly lower than the normal (Stickle et al., 1989). More mobile and migratory species tend to avoid areas with DO concentrations below about 3 mg/l (Keller and Squibb, 1992). The low DO levels often detected at the Site have affected the distribution of species that are present. Thus, DO is a major stressor to the Site ecosystem.

4.1.3.3 Chemicals in Sediments and Surface Water

As indicated in Section 4.1.1, the problem of hazardous chemicals in sediments and surface water at the Site has been the focus of much concern in recent years. These chemicals are considered to be stressors to the Site ecosystem. The distributions and concentrations of various chemicals at the Site are discussed in detail in Section 4.3 (Selection of Chemicals of Potential Concern), and throughout the remainder of this screening-level ERA.

4.2 Characterization of the Ecological Community

The objective of Section 4.2 is to characterize the local ecosystem in terms of both physical characteristics and ecosystem structure. Hydrography, surrounding land use, and the presence and extent of aquatic and wetland habitats are among the physical characteristics evaluated. Ecosystem

structure is described in terms of vegetative cover types, sizes and types of habitats, types and estimated abundances of major ecological receptor groups, and the presence of endangered and/or threatened species.

4.2.1 Data Acquisition and Evaluation

Historical data on the physical characteristics and ecology of the Site and the tidal Passaic River were compiled and evaluated. The sources of data include available technical reports, published and unpublished scientific data, and habitat and environmental resource maps and photographs. These data were used to supplement data collected during a Habitat Survey and Finfish and Benthic Invertebrate Survey, both of which were conducted by ChemRisk ecologists in August, 1994. The methods and results of these surveys are presented in two reports entitled *Evaluation of Aquatic and Terrestrial Habitats Within the Passaic River Study Area* (ChemRisk, 1995a), and *Finfish and Benthic Invertebrate Survey of the Passaic River Study Area* (ChemRisk, 1995b). These reports are provided in Appendix E and F, respectively.

In addition to Site-specific data and historical data collected from the tidal Passaic River, regional studies that include data on the Newark Bay Estuary and other relevant areas within the NY/NJ Harbor Estuary were evaluated and, if appropriate, used to support the data on potential distributions of key migratory species that potentially occur at the Site.

Data were evaluated with respect to their scientific integrity. Evaluated criteria included acceptability of the collection and analytical methodologies, appropriateness of the sampling plan and technique(s), and location and date of sampling. Because the historical investigations of the fish and benthic invertebrate communities in the tidal Passaic River are highly qualitative in nature, and because more recent data have been collected for these organisms, the historical investigations are discussed only in terms of the species identified and their abundance. Sampling methods do not affect the quality of surveys intended only to identify organisms that are present in a system. Therefore, other than the date and location of sample collections, collection protocols are not a key factor in determining the validity of these investigations. For the most part, however, historical sampling methodologies for fish and benthic invertebrates were consistent with those used in the August, 1994 surveys.

4.2.2 Physical Characteristics

4.2.2.1 Land Use and Human Development

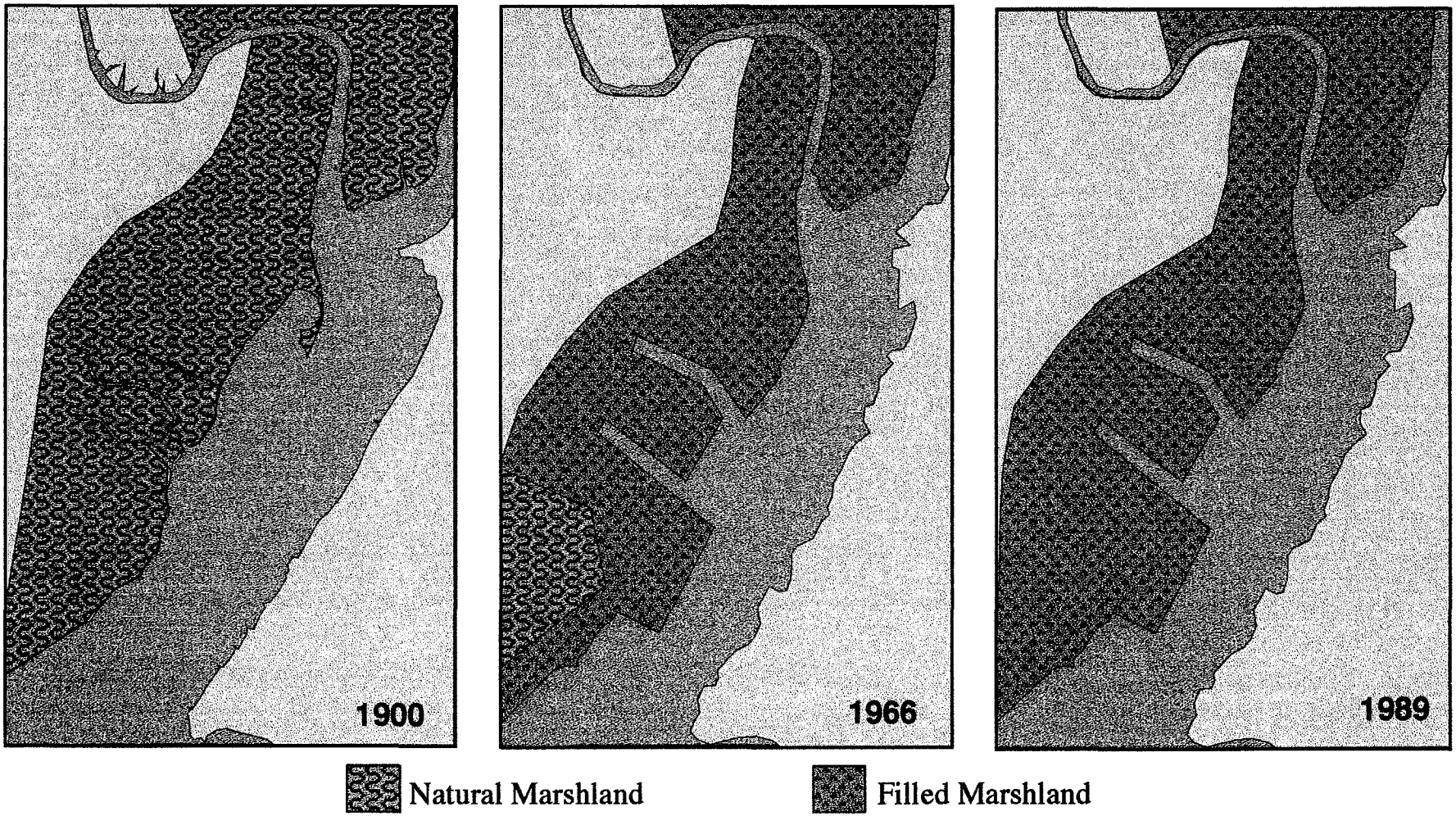
The physical characteristics of the Site are described in detail and illustrated in a complete photographic record of the Site in Appendix E. As discussed in Section 1.0, the Site has a long history of industrialization, dating back more than two centuries (Meyers, 1945; Cunningham, 1966a,b; Brydon, 1974). Land use along the lower Passaic River, extending south of the Dundee Dam and including the Site, is dominated by high-density commercial and industrial development.

The left bank of the Site (looking upriver from the lower Site boundary) is almost fully developed, consisting of active or abandoned commercial and/or industrial properties. Similarly, the right bank of the Site (looking upriver) is comprised primarily of abandoned industrial properties and railroad lines. A highly developed network of highways and local streets exist on lands adjacent to the Site and several bridges cross over the Site. In addition, a large network of municipal and industrial outfalls drain into the lower Passaic River. These include CSOs, stormwater outfalls, and POTWs outfalls (Mueller et al., 1982).

As depicted in Figure 4-1, nearly all of the wetlands that once existed in the lower Passaic River have been filled (i.e., reclaimed) and, thus, eliminated, with more than 7,500 acres being reclaimed just since the 1940s (ACOE, 1987). Wetlands reclamation has resulted in a large increase in available land mass for industrial development. In addition, concurrent with industrial and commercial development adjacent to the River, much effort has been directed toward stabilizing river banks at the Site, which has resulted in substantial loss of shallow water habitats and wetlands.

In addition to wetlands reclamation and shoreline alterations that have occurred over the past century, the lower Passaic River has been dredged periodically to develop and maintain navigation channels for commercial boat traffic (ACOE, 1988). Since 1919, over 10,000,000 cubic yards of sediment have been removed from the River as the result of greater than 25 dredging events (ACOE, 1988). The depth alterations that have been produced within the River by navigational dredging have fragmented and removed much of the available shallow water habitat, and altered the hydrology of the Site.

Figure 4-1. Man-Made Alterations to the Estuarine Habitats and Shoreline of the Lower Passaic River and Newark Bay, New Jersey from 1900 to 1989



Adapted from Squires and Barclay (1990)

The net result of human development adjacent to the Site has been the elimination of nearly all terrestrial and wetland habitats at or adjacent to the Site, and the severe degradation of the riverine (aquatic) habitat. These changes have been previously documented by several authors (Brydon, 1974; Squires and Barclay, 1990; ERM, 1990; Zdepski, 1992; Crawford et al., 1994, 1995), and are discussed further in Section 4.2.3.

4.2.2.2 Hydrology

The lower Passaic River, including the Site, is a tidal tributary of Newark Bay. The River is influenced by semi-diurnal tides for approximately 17 miles, extending from Dundee Dam downstream to its confluence with Newark Bay. The mean tidal range (difference in height between mean high water and mean low water) at the New Jersey Turnpike Bridge (approximately 1.5 miles upstream from Newark Bay, at Site mile 1.7) is 5.1 feet (NOAA, 1972) with a mean tidal elevation of 2.5 feet (NOAA, 1972). The mean spring tide range (average semi-diurnal range occurring during the full and new moon periods) is 6.1 feet.

Saline to brackish water conditions exist throughout the Site. In August, 1994, salinities ranged from 6 ppth to 23 ppth. Salinities are nearly similar over most of the Site (about 13 to 23 ppth), with the exception of the area near the upstream boundary of the Site (Mile 6), where salinities are lower (about 6 to 9 ppth). The cross-sectional average river velocity due to freshwater flow in the Site is approximately 1 foot per second and a typical maximum tidal velocity is about 3 feet per second (ACOE, 1987). The range in salinities, and effects of semi-diurnal tides influence the species that comprise the Site ecosystem, as discussed below.

4.2.3 Ecological Habitat Characterization

A habitat survey for the Site was conducted by ChemRisk ecologists in August, 1994 (ChemRisk, 1995a). The purpose of the survey was to delineate and evaluate existing habitats at the Site, and to supplement historical reports. The survey was conducted during the summer growing season, when the diversity and abundance of organisms are expected to be the greatest. This was also the time when marsh grasses and other wetland vegetation, as well as submerged aquatic vegetation (SAV) were likely to be fully grown.

Available ecological habitats at the Site were identified and individually evaluated for their suitability as primary foraging or roosting areas for birds or mammals, as well as nursery grounds for aquatic organisms. The results of historical investigations regarding the industrial and urban development in the area were also considered in evaluating the quantity and quality of available habitats.

4.2.3.1 Terrestrial Habitats

As depicted in photographs taken during the August, 1994 habitat survey, as well as in aerial photographs taken in June, 1994 (see Appendix E), there is little, if any, suitable habitat adjacent to the Site to support terrestrial wildlife, particularly birds or mammals. Although shoreline vegetation is present in some areas, it is generally limited to narrow buffer zones (< 20 feet in width) of grasses, woody perennials, such as *Phragmites* sp. or *Artimesia* sp., or narrow bands of trees such as willows (*Salix* sp.), maples (*Acer* sp.), and Sumac (*Rhus* sp.), that are left for aesthetics and shoreline (bank) stabilization (Appendix E; ACOE, 1987). These areas are usually bordered by industrial facilities, highways, or parking lots. They are typically very fragmented, and therefore unlikely to support significant populations of wildlife. The ACOE (1987) concluded that the habitat diversity along the lower Passaic River, especially near the city of Newark, NJ was low, with very limited food and cover available.

4.2.3.2 Aquatic and Wetland Habitats

As discussed in Section 4.1.1.1, nearly all of the wetlands that were once present at the Site have been reclaimed, while the small amounts of wetlands that remain have been significantly altered by a variety of human activities (Squires and Barclay, 1990). Based on the results of the August 1994 habitat survey, as well as earlier surveys, it is evident that the Site lacks sufficient wetlands (i.e., marsh) habitat to provide adequate nursery or foraging areas for most aquatic species (Appendix E; ACOE, 1987; USFWS, 1981). Although areas of aquatic vegetation, including saltmarsh cordgrass (*Spartina alterniflora*) do exist, they are limited in size and occur sporadically throughout the Site.

In total, 73 percent of the shoreline of the Site is comprised of either bulkheads or riprap (61 percent and 12 percent, respectively) (Table 4-1; Appendix E). Three localized areas were identified as having conditions (i.e., vegetation, exposed mudflats) suitable to provide cover for some aquatic organisms; however, the largest of these was estimated to be approximately 0.3 acres in size, and combined, they comprise less than one acre. Due to severe size limitations, it is unlikely that these areas would attract or support significant populations of resident or migratory finfish species (Boesch and Turner, 1981; ACOE, 1987). Furthermore, there are no habitats that appear suitable for aquatic or semi-aquatic mammals (i.e., muskrats, mink etc.) at the Site.

Available aquatic habitats at the Site are also limited as a result of the intense urbanization of the surrounding area. Although the River provides a passageway for fish movements, and residence for some aquatic organisms, the conditions of the habitat at the Site are extremely poor and degraded, primarily due to poor sediment and water quality. In addition, only about 18 percent of the shorelines of the Site contain intertidal mudflats (Table 4-2). In the absence of wetland (i.e., marsh) habitats, mudflats are the primary shallow water habitats that can provide cover for burrowing crustaceans or mollusks, and food sources for predators of these organisms. However, because of their limited size and sediment quality, as well as the absence of associated marsh habitat, it is unlikely that the mudflats within the Site provide sufficient quality habitat to support significant populations of organisms.

4.2.4 Evaluation of Ecological Community Data

A finfish and benthic invertebrate survey of the Site was conducted by ChemRisk ecologists in August, 1994 (ChemRisk, 1995b). The results of the survey, as well as historical ecological investigations conducted within the tidal Passaic River, have reported that the Site supports a limited number of both freshwater and estuarine species (Appendix F; PAS, 1982; ACOE, 1987). As discussed in Section 4.1.2.2, saline to brackish water conditions exist throughout the Site with the exception of the area near the upstream boundary of the Site (Mile 6), where salinities are lower, approaching freshwater conditions. This gradient affects the distributions of species at the Site, as discussed below.

Table 4-1. Shoreline Features at the Site

	Point No Point Reach				Harrison Reach				Newark Reach			
	Right Bank		Left Bank		Right Bank		Left Bank		Right Bank		Left Bank	
	Approx. ft	Percent of Total	Approx. ft	Percent of Total	Approx. ft	Percent of Total	Approx. ft	Percent of Total	Approx. ft	Percent of Total	Approx. ft	Percent of Total
Bulkhead (Metal, stone, or wood)	4500	67%	4000	60%	3000	26%	6000	53%	6700	87%	4500	58%
Riprap	1500	22%	2550	38%	1000	9%	1250	11%	1000	13%	NO	NO
Mixed (a)	700	10%	NO	NO	2400	21%	3400	30%	NO	NO	3200	42%
Aquatic Vegetation	NO	NO	150	2%	5000	44%	750	7%	NO	NO	NO	NO
Total Shoreline (ft.)	6700		6700		11400		11400		7700		7700	

	Kearny Reach				Arlington Reach (b)				Cumulative Total for Study Area					
	Right Bank		Left Bank		Right Bank		Left Bank		Right Bank		Left Bank		Total Shoreline (c)	
	Approx. ft	Percent of Total	Approx. ft	Percent of Total	Approx. ft	Percent of Total	Approx. ft	Percent of Total	Approx. ft	Percent of Total	Approx. ft	Percent of Total	Approx. ft	Percent of Total
Bulkhead (Metal, stone, or wood)	4200	81%	5200	100%	NO	NO	680	100%	18400	58%	20380	64%	38780	61.2%
Riprap	500	10%	NO	NO	NO	NO	NO	NO	4000	13%	3800	12%	7800	12.3%
Mixed (a)	NO	NO	NO	NO	680	100%	NO	NO	3780	12%	6600	21%	10380	16.4%
Aquatic Vegetation	500	10%	NO	NO	NO	NO	NO	NO	5500	17%	900	3%	6400	10.1%
Total Shoreline (ft.)	5200		5200		680		680		31680		31680		63360	

Source: ChemRisk, 1995a
NO = Not Observed
a. "Mixed" refers to a mixture of rip-rap and aquatic vegetation.
b. Refers only to that portion of Arlington Reach included in the Passaic River Study Area.
c. Total Shoreline is equivalent to the sum of the linear distance both right and left banks of the River.

Table 4-2. Estimated Occurrence of Shoreline Vegetation and Mudflats at the Site

	Shoreline Vegetation (a)				Mudflats (b)			
	Right Bank		Left Bank		Right Bank		Left Bank	
	Approx. ft.	Percent of Total (c)	Approx. ft.	Percent of Total (c)	Approx. ft.	Percent of Total (c)	Approx. ft.	Percent of Total (c)
Point No Point Reach	1,000	15%	1,500	22%	NO	NO	1,500	22%
Harrison Reach	5,000	44%	3,000	26%	4,000	35%	2,500	22%
Newark Reach	500	6%	1,500	19%	750	10%	NO	NO
Kearny Reach	2,000	38%	500	10%	1,000	19%	1,000	19%
Arlington Reach (d)	680	100%	500	74%	NO	NO	680	100%
Total For Study Area	9,180	29%	7,000	22%	5,750	18%	5,680	18%

Source: ChemRisk, 1995a
NO=Not Observed
a. Shoreline vegetation refers to areas containing shrubs and trees. Large grassy areas may also be included. Areas of shoreline vegetation may co-occur with other features; for example, a stand of trees or shrubs may be present regardless of whether the bank is bulkhead, riprap, or aquatic vegetation. Therefore, these features are not included in estimate of total shoreline.
b. Mudflats refers to areas where mud substrate is exposed at low tide. Similar to shoreline vegetation, areas of mudflats may co-occur with other features, therefore, they are not included in estimate of total shoreline.
c. Represents the percent of the river bank in the indicated reach along which the feature occurs.
d. Refers only to that portion of Arlington Reach located within the Study Area.

4.2.4.1 Plankton

Phytoplankton and zooplankton communities within the tidal Passaic River were surveyed by Princeton Aqua Science (PAS) in the fall of 1981. Periphyton (attached microalgae) communities were evaluated as part of this survey in the fall of 1981 and the spring of 1982, and are discussed in this Section. Data were collected from four locations within the Site and one location upstream of the Site, but within the tidal portion of the River (Figure 4-2). Plankton samples were collected from the water column using 63 μm mesh plankton net samplers. Periphyton (scrape) samples were collected from artificial substrates which were deployed in the lower portion of the water column and allowed to colonize for three weeks. The collection methods employed in this survey are consistent with standard scientific collection procedures. The results of the investigation are, therefore, applicable for use in the screening-level ERA.

Phytoplankton data were collected from four locations within the Site in the fall of 1981 (PAS, 1982). The species list, as compiled from the four samples, is provided in Table 4-3. The results of the survey suggest that the fall phytoplankton community is dominated by diatoms (bacillariophyta), primarily pennate diatoms, followed by centrates and naviculoids. Blue-green algae (cyanophyta), green algae (chlorophyta), and euglenoids (euglenophyta) were also present, but generally comprised less than about 10 percent of the phytoplankton biomass. PAS (1982) concluded that the phytoplankton assemblage in the River is generally indicative of a pollution-stressed environment. The dominance of diatoms in the community is consistent with fall blooms of these organisms that are characteristic of waterways in the north temperate zone of North America (Day et al., 1989). It is likely that the species composition does not vary substantially throughout the year, however, the dominance in biomass will shift between seasons. For instance, either diatoms, blue-green, and/or green algae may dominate the biomass in the winter and through the spring blooms. However, blue-green algae characteristically bloom in late summer, and may dominate the biomass during this period of the year (Day et al., 1989).

The results of the periphyton survey are presented in Table 4-4. The species assemblage was comprised exclusively of diatoms. Dominant genera (in terms of biomass) include *Navicula*, *Nitzschia*, *Fragilaria*, *Asterionella*, and *Cyclotella*. Similar to the phytoplankton assemblage, the periphyton were also dominated by pollution-tolerant species (PAS, 1982). In the fall, the

Figure 4-2. Plankton and Periphyton Sampling Locations in the Tidal Passaic River, PAS, 1981 - 1982

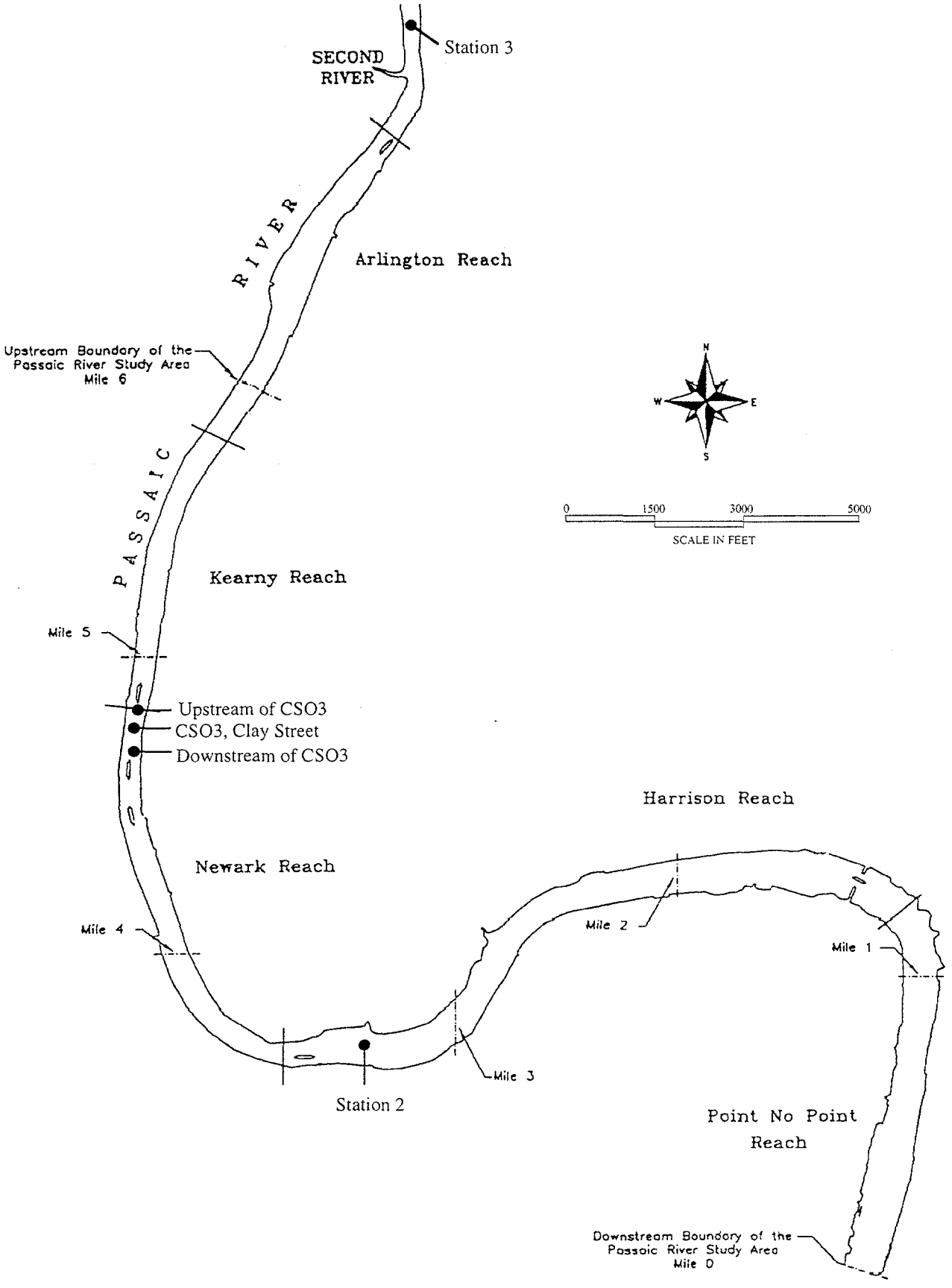


Table 4-3. Phytoplankton and Zoolplankton Identified in the Tidal Passaic River, Fall, 1981

Taxon	Taxon
Phytoplankton (a)	**Phytoplankton (cont'd)**
Cyanophyta	Bacillariophyta (cont'd)
coccoid	M. nummuloides
Microcystis flos-aquae	M. varians
Oscillatoria sp.	Meridion circulare
Chlorophyta	Navicula sp.
colonial	Nitzschia sp.
Ankistordesmus convoolutus	Pinnularia sp.
A. falcatus	Plagiotropis lepidoptera
Chlamydomonas	Rhoicosphenia curvata
Closterium sp.	Rhopalodia sp. (broken)
Coelastrum microporum	Skeletomena costatum
Cosmarium sp.	Surirella sp.
Pediastrum duplex	Surirella (side)
P. duplex var gracillimum	S. ovata
P. simplex	Synedna ulna
P. simplex var duodenarium	Triaceratium alternans
Scendesmus sp. S. longus	spiny
S. quadricauda	Mallomonas sp.
Spirogyra sp.	Dinobryon
Staurastrum	
Euglenophyta	**Zooplankton (b)**
Euglena sp.	Rotifera
Trachelomonas sp.	Keratella
T. euchlora	Rotifera sp.
Bacillariophyta	Filinia
centrate	Brachionis
pennate	Protozoa
naviculoid	Ceratium
Achnanthes linearis	Arthropoda, Cladocera
Amphora ovalis	Chydorus
Asterionella formosa	Alona
Cocconeis placentula	Arthropoda, Copepoda
Coscinodiscus sp.	Paracyclops
C. lacustris	Argulus nauplii
C. rothii	
Cyclotella glomerata	
C. menegheniana	
Cymatopleura solea	
Cymbella sp.	
Diatoma vulgare	
Diploneis sp.	
Ditylum sp.	
D. brightwelli	
Entomoneis sp.	
Fragilaria sp.	
F. construens	
F. crotonensis	
Gomphonema sp.	
Gomphonema (slide)	
Gyrosigna/Pleurosigma	
G. fasicola (?)	
Melosira sp.	

Source: PAS, 1982
a. Phytoplankton were collected within and upstream of the Site
b. Zooplankton were collected upstream of the Site

Table 4-4. Periphyton Identified in the Tidal Passaic River, Fall, 1991 and Spring, 1982 (a)

Taxon	Taxon
Bacillariophyta	Bacillariophyta (cont'd)
Achnanthes sp.	Navicula sp.
A. clevei	N. capitata
A. exigua	N. cryptocephala
A. lanceolata	N. cuspidata
A. lanceolata var dubia	N. exigua
A. linearis	N. integra
A. macrocephala	N. lanceolata
A. minutissima	N. mutica
Amphora coffeiformis	N. pupula
Amphora ovalis	N. pusilla
Asterionella formosa	N. pygamea
Caloneis sp. #1	N. radiosa
C. bacillum	N. rhynchocephala
Cocconeis placentula	N. secreta
C. placentula var lineata	N. tripunctata
Coscinodiscus lacustris	Nitzschia sp.
C. rothii	N. acicularis
Cyclotella menegheniana	N. amphibia
C. stelligera	N. dissipata
Cymbella sp	N. filiformis
C. affinis	N. hungarica
C. minuta	N. longissima
Cymatopleura solea	N. palea
Diatoma tenue var elongatum	N. parvula
D. vulgare	N. sigma
Entomoneis paludosa	N. tryblemella
Epithemia adnata	Pinnularia sp.
Eunotia spp.	P. stomataphora
E. curvatus	Rhoicosphenia curvat
E. praerupta	Rhopalodia sp.
Fragilaria sp.	R. gibba
F. construens	Skeletonoema costatu
F. crotonensis	Stauroneis anceps
F. leptostauron	Steneroptera intermed
F. pinnata	Stephanodiscus astrae
F. vaucheriae	Surirella sp.
Frustulia rhomboides var amphipleuroides	S. angustata
F. rhomboides var saxonica	S. ovalis
Gomphonema sp.	S. ovata
G. acumentum	Synedra sp.
G. angustatum	S. acus
G. olivaceum	S. delicatissima
G. parvulum	S. fasciculata
G. sphaerophorum	S. incisa
G. truncatum	S. radians
Gyrosigma sp.	S. rumpens
Hantzschia amphioxys	S. ulna
Melosira granulata	Tabellaria fenestrata
M. nummuloides	Thalassiosira fluviatil
M. varians	
Meridion circulare	
M. circulare var. constrictum	

Source: PAS, 1982

a. Periphyton were collected within and upstream of the Site

periphyton assemblage was dominated by *Cyclotella meneghenia* and *Nitzschia spp.* In May, *Navicula spp.* were dominant, followed by a mixture of species including *Nitzschia spp.*, *Asterionella formosa*, *Fragillaria consturens*, *Surirella spp.*, and *Synedra sp.*

Zooplankton data were not collected from the Site, but were collected upstream of the Site, in the tidal portion of the River (Table 4-3). Data collected in the fall of 1981 indicate that rotifers, particularly the genera *Keratella*, *Rotifera*, and *Brachionis*, dominate the zooplankton community in the tidal portion of the River. Together, these three genera accounted for greater than 73 percent of the zooplankton biomass. In total, rotifers accounted for 74 percent of the zooplankton biomass, followed by the protozoan *Ceratium sp.* (13 percent), and arthropods (13 percent), including equal proportions of cladocerans (*Chydorus sp.* and *Alona sp.*), and calanoid copepods (*Paracyclops sp.* and *Argulus nauplii*).

4.2.4.2 Benthos

In general, the results of benthic invertebrate surveys conducted in the Newark Bay Estuary, including the tidal Passaic River, suggest that benthic diversity is very low (ACOE, 1980; PAS, 1982; Berg and Levinton, 1985; Cerrato and Bokuniewicz, 1986; Cerrato, 1986; Cristini, 1991). For example, a survey conducted within and upstream of the Site in the tidal Passaic River in 1981 indicated that the benthic invertebrates present were limited to those species capable of surviving extremely poor water quality conditions (PAS, 1982). In addition, the diversity of invertebrates was extremely low, and in some instances there were no organisms found in sediment samples. When found, nearly 100 percent of the invertebrate biomass was dominated by the oligochaetes *Limnodrilus sp.* and *Lumbricus sp.* It should be noted that the sampling locations for this study were located adjacent to CSOs, which may explain the extremely low diversity.

Table 4-5 lists the benthic invertebrate species that have historically been reported to occur in the tidal Passaic River. The current benthic community was characterized as part of the biological survey that was performed by ChemRisk ecologists in August, 1994 (Appendix F). The results indicate that the benthic invertebrate community at the Site is dominated by polychaete (primarily *Streblospio benedicti* and *Hypaniola grayi*) and oligochaete worms (primarily *Limnodrilus hoffmeisteri* and Tubificidae sp.). Together, these organisms comprise 68 to 98 percent of all organisms observed at various sampling locations throughout the Site. Other species observed at

Table 4-5. List of Benthic Inveterbrates Identified from the Tidal Passaic River

Taxon	Passaic River Study Area	Reference
Annelida		
Oligochaeta		
Limnodrilus hoffmeisteri	x	ChemRisk, 1995b
Limnodrilus sp.	x	PAS, 1982
Lumbriculus sp.	x	PAS, 1982; ACOE, 1987
Naidae sp.	x	PAS, 1982
Polycheata		
Eteone heteropoda	x	ChemRisk, 1995b
Hypaniola grayi	x	ChemRisk, 1995b
Laeonereis culveri	x	ChemRisk, 1995b
Scolecolepides viridis	x	ChemRisk, 1995b
Scoloplos sp.	x	ChemRisk, 1995b
Streblospio benedicti	x	ChemRisk, 1995b
Arthropoda		
Crustacea		
Asellus sp.	x	ACOE, 1987
Callinectes sapidus	x	Hauge, 1993; ChemRisk, 1
Cyathura polita	x	ChemRisk, 1994
Edotea triloba	x	ChemRisk, 1994
Leucon americanus	x	ChemRisk, 1994
Rithropanopeus harrisii	x	ChemRisk, 1994
Insecta		
Chironomidae		ACOE, 1987
Cochagriidae		ACOE, 1987
Procladius sp	x	ChemRisk, 1995b
Mollusca		
Bivalvia		
Veneroida	x	ChemRisk, 1995b
Mytilopsis leucophaetata	x	ChemRisk, 1995b

most stations included the crustaceans *Cyathura polita*, *Rhithropanopeus harrisii*, and *Leucon americanus*. Insects and mollusks were observed infrequently and in relatively low numbers throughout the Site. In general, polychaetes were the dominant species at stations located in the more downstream (i.e., saline) portion of the Site, while oligochaetes were predominant in areas with lower salinities near the upstream end of the Site.

The benthic invertebrate community within the Site is largely influenced by the industrial and urban nature of the surrounding area. In general, the species composition, diversity, and abundance at the Site are characteristic of a degraded estuarine environment (Appendix F). For example, Shannon-Weaver Diversity Index (H′) values based on the benthic invertebrate samples collected during the August 1994 survey ranged from 0.236 to 1.66, indicating that the biological diversity of the benthic invertebrate community of the Site is low. Diversity values below 2 are generally considered to be indicative of pollution stress (Stainken, 1984).

In addition to the organisms identified in the benthic invertebrate survey, dip net sweeps taken in August, 1994 provided a qualitative evaluation of the species present in the limited vegetated shoreline areas of the River. Grass shrimp (*Palaemonetes pugio*) and the amphipod, *Ampelisca abdita*, were observed in the dip nets sweeps. Large numbers of amphipods and mud crabs were also observed in leaf litter inadvertently collected in gill nets that were set for the finfish survey. Blue crabs were also collected in all but two of the gill net deployments. Blue crabs are commonly reported as an abundant species throughout the tidal Passaic River and the Newark Bay Estuary, and are considered to be dominant macrofaunal species in the Estuary (Belton et al., 1983, 1985; Hauge et al., 1990, 1993). Physical data on the blue crabs that were captured are provided in Appendix F.

4.2.4.3 Finfish

Similar to the plankton and benthos at the Site, it has been demonstrated that the fishes of the tidal Passaic River are dominated by pollution-tolerant species (USFWS, 1981; ACOE, 1987). Fish surveys conducted in 1973 identified 24 species of fish in the tidal Passaic River (Festa and Toth, 1976). Twenty-three species of fish were observed during various investigations from June 1977 to March 1979 (Santoro et al., 1980), however a survey conducted in 1981 reported 41 species of fish (ACOE, 1987).

Table 4-6 provides a list of fish species that have historically been identified in the tidal Passaic River (PAS, 1982; Belton et al., 1985; ACOE, 1987). It should be noted that most of the species listed in Table 4-6 were individual fish that were captured during only one sampling event, and at only one location in the tidal portion of the River (see PAS, 1982; Belton et al., 1985; ACOE, 1987). Therefore, the actual diversity of fish species in the tidal Passaic River is likely much lower than that portrayed in Table 4-6.

Fish communities in the River are comprised of a mixture of marine, estuarine, and freshwater species (Woodhead, 1991). Resident estuarine species appear to be primarily limited to the mummichog (*Fundulus heteroclitus*) (PAS, 1982; ACOE, 1987; Appendix F). Migratory species such as striped bass, american eel, and white perch appear to be relatively common in the tidal Passaic River (USFWS, 1981; ACOE, 1987). Common freshwater species reported in the River include carp, goldfish, golden shiner, and pumpkinseed (ACOE, 1987; PAS, 1982; USFWS, 1981).

Based on the data collected in the August, 1994 fish survey, mummichog (*Fundulus heteroclitus*) appear to be the dominant fish species present at the Site. This is consistent with the results of earlier studies. ACOE (1987) reported that mummichog comprised 94 percent of the fish community in the tidal Passaic River. Similarly, mummichog were the only species of fish found by PAS (1982) within the Site. Ichthyological Associates found that mummichog accounted for more that 50 percent of the total density of fish impinged on intake screens at POTW in the tidal Passaic and Hackensack Rivers, and averaged 66 percent of the total number of individuals captured in otter trawls from these areas (Berg and Levinton, 1985).

4.2.4.4 Other Organisms

The only mammals observed in the Site during the August 1994 survey (Appendix E) were rats seen along the bulkheads and shorelines of the River. This is consistent with the conclusions of the ACOE (1987) which reported that terrestrial species along the lower Passaic River are limited to human-tolerant species commonly found in urban environments. In addition, there are no apparent habitats suitable for aquatic or semi-aquatic mammals (i.e., muskrats, mink etc.) within the Site;

Table 4-6. List of Fish species Identified from the Tidal Passaic River

Species	Scientific Name	References
Alewife	*Alosa pseudoharengus*	Berg & Levinton, 1985; ACOE, 1987; USFWS, 1981
American eel	*Anguilla rostrata*	ACOE, 1987; Berg & Levinton, 1985; Belton et al., 1982-1990; USFWS, 1981
American shad	*Alosa sapidissima*	ACOE, 1987; USFWS, 1981
Atlantic menhaden	*Brevoortia tyrannus*	Belton et al., 1982; USFWS, 1981
Atlantic silverside	*Menidia menidia*	ACOE, 1987; ChemRisk 1995b
Banded killifish	*Fundulus diaphanus*	USFWS, 1981
Bay anchovy	*Anchoa mitchilli*	Berg & Levinton, 1985; ACOE, 1987
Black crappie	*Pomoxis nigromaculatus*	ACOE, 1987
Blacknose dace	*Rhinichthys atratulus*	USFWS, 1981
Blueback herring	*Alosa aestivalis*	Berg & Levinton, 1985; ACOE, 1987; USFWS, 1981
Bluegill	*Lepomis machrochirus*	ACOE, 1987; USFWS, 1981
Brown bullhead	*Ameirurus nebulosus*	ACOE, 1987; Belton et al., 1985; USFWS, 1981
Carp	*Cyprinus carpio*	ACOE, 1987; ChemRisk, 1995b; Belton et al., 1982, 1985, 1993; 1990; USFWS, 1981
Channel catfish	*Ictalurus punctatus*	USFWS, 1981
Common shiner	*Luxilus cornutus*	USFWS, 1981
Gizzard shad	*Dorooma cepedianum*	USFWS, 1981
Gobies	*Gobiidae sp.*	Berg & Levinton, 1985
Golden shiner	*Notemigonus crysoleucas*	USFWS, 1981
Goldfish	*Carassius auratus*	ACOE, 1987; Belton et al., 1985; USFWS, 1981
Largemouth bass	*Micropterus salmoides*	Berg & Levinton, 1985; Belton et al., 1982; USFWS, 1981; ACOE, 1987
Mummichog	*Fundulus heteroclitus*	Berg & Levinton, 1985; ACOE, 1987; PAS, 1982; ChemRisk, 1995b; USFWS, 1981
Northern pipefish	*Syngnathus fuscus*	ACOE, 1987
Pumpkinseed	*Lepomis gibbosus*	Belton et al., 1982; ACOE, 1987; USFWS, 1981
Red Hake	*Urophycis chuss*	ACOE, 1987
Satinfin shiner	*Cyprinella analostana*	USFWS, 1981
Silver hake	*Merluccius bilinearis*	Berg & Levinton, 1985; ACOE, 1987
Silvery minnow	*Hybognathus nuchalis*	USFWS, 1981
Spot	*Leiostomus xanthurus*	ACOE, 1987
Striped bass	*Morone saxatilis*	Hauge, 1993; ACOE, 1987;Belton et al., 1982, 1983, 1993; 1990; USFWS, 1981
Threespine stickleback	*Gasterosteus aculeatus*	ACOE, 1987
Tidewater silverside	*Menidia peninsulae*	ACOE, 1987; USFWS, 1981
Tomcod	*Microgadus tomcod*	Berg & Levinton, 1985; ACOE, 1987
White catfish	*Ameiurus catus*	Belton et al., 1983
White sucker	*Catostomus commersoni*	ACOE, 1987; USFWS, 1981
White perch	*Morone americana*	Belton et al., 1982, 1983; ACOE, 1987; Berg & Levinton, 1985; USFWS, 1981

the limited wetland areas identified within the Site are too small to support significant populations of such organisms. For these reasons, a terrestrial food web is not considered further in this screening-level risk assessment.

Similarly, there are no nesting areas for aquatic birds within the Site. The extent of available wetlands habitat (that would be necessary for roosting) at the Site is negligible (i.e., less than one hundredth of one percent), compared to that available locally. In addition, wading birds are far more likely to feed in the large, relatively undisturbed wetlands near their roosting sites than along the banks of the highly industrialized lower Passaic River for several reasons.

First and foremost and, in contrast to the Site, there are a number of extensive, high-quality roosting and foraging habitats in the NY/NJ Harbor Estuary, including over 8,000 acres in the local environs (Squires and Barclay, 1990). These areas not only provide high quality roosting habitat, but also provide a much more diverse and abundant assemblage of prey for wading birds. Like other organisms, birds have adapted behaviors that minimize their maintenance energy costs, since inefficiency can place individuals at a competitive disadvantage (Recher and Recher, 1969; Greig et al., 1983; MacLean, 1986). To that end, birds employ the following key behavioral adaptations to foraging strategies:

- minimize respiratory energy loss, by limiting flight frequency and distance within a foraging area and between foraging area and roost;
- maximize foraging success by selecting areas with abundant prey; and,
- avoid potential disturbances (by humans or predators) and the energy expenditures associated with defense, by foraging in areas that are remote or provide cover.

Given the absence of appropriate habitat for wading birds at the Site, and the low diversity of prey (for birds) in the industrialized Passaic River, it is highly unlikely that bird populations would obtain a significant proportion of their diets in the immediate vicinity of the Site. Rather, it is more likely that relatively few individuals of wading birds forage intermittently and seasonally on the limited number of mudflats that comprise the Site. Because both exposure duration at the Site, and the fraction of prey that would be consumed from the Site, are very low for the wading bird

populations relative to other sites in the NY/NJ Harbor Estuary, the resulting risks from chemical stressors that would be calculated in a risk assessment for the Site would be negligible. For these reasons, a quantitative risk assessment for birds is not warranted at the Site.

4.2.4.5 Threatened or Endangered Species

There are no state or federal rare, threatened, or endangered species that are known to inhabit the Site (USFWS, 1981; NOAA, 1993; NJDEP, 1995). Therefore, threatened or endangered species are not considered further in this screening-level risk assessment.

4.2.5 Food Web Evaluation

The sequence of organisms through which energy may move within an ecological community is customarily called a food chain. In most communities, the many possible food chains are so complexly intertwined, that together they form a community food web. Certain basic characteristics are present in all food webs; every food web begins with the autotrophic organisms (e.g., green plants) that are the primary producers for the community and ends with the decomposers (e.g., bacteria, fungi) which release simple substances reusable by the primary producers (Odum, 1972). The links between the producers and the decomposers are more variable and may include primary, secondary, and tertiary consumers. The successive stages of a food web represent the trophic levels of the community (Odum, 1972; Keeton, 1980). In addition to energy, nutrients and other chemicals are transferred through different trophic levels. The extent of loss or accumulation of energy and chemicals at each successive trophic level is quite variable, but is largely a function of the bioenergetics of the organism and the physicochemical properties of the chemical. The food web of the Site is discussed below.

4.2.5.1 Identification of Key Aquatic Organisms

Consistent with the IWP and EPA guidance (1989c, 1992a, 1994a), "key organisms" in a food web include: (a) resident organisms subject to the greatest exposure to contaminated sediments and water; (b) species considered to be essential to, or indicative of, the normal functioning of the existing habitat; and (c) federal or state threatened or endangered species. As stated in Section

4.2.4.5, there are no known rare, threatened, or endangered species that inhabit the Site. The key species are thus limited to dominant organisms at each trophic level (resident and migratory) that are subject to the greatest potential exposure to contaminated sediments and water.

At the primary producer level, the key organisms are phytoplankton, since macrophytic vegetation is not present throughout most of the Site. At the primary consumer level, zooplankton, particularly rotifers, protozoans, cladocerans, and calanoid copepods are predominant in the water column, and benthic invertebrates, particularly polychaete and oligochaete worms are predominant in sediments. Phytoplankton and zooplankton are considered in the risk assessment as generic groups (i.e., non-species specific) at their respective trophic levels, since the mechanisms for exposure to and uptake of chemicals for all taxa within these groups are considered to be essentially the same (Clayton et al., 1977).

At the secondary consumer level, the mummichog is predominant, and appears to comprise most of the biomass within the Site. It is readily apparent from the historical data that the mummichog is essential to the normal functioning of the existing Site habitat. Although other forage fish species, such as the carp and Atlantic silverside may be present at the Site, the mummichog is clearly the most widespread and dominant forage fish in the River and, thus, is the appropriate indicator species for this trophic level.

Tertiary consumers at the Site include the blue crab (large omnivorous crustacean) and large predatory fish, such as striped bass, bluefish, and american eel. The blue crab, like the mummichog, has clearly been shown to be essential to the normal functioning of the existing Site habitat. Of the predatory fish species that forage at the Site, the striped bass appears to be most dominant in the tidal Passaic River, and the American eel is also seasonally present in substantial numbers. Both of these fish are considered to be key aquatic organisms at the Site. Based on the results of historical surveys, bluefish do not appear to be present very often in the tidal Passaic River and, therefore, are not considered to be a key organism at the Site.

4.2.5.2 Construction of Simplified Food Web

Because of the low diversity of species that occur at the Site, and the limited number of key organisms that have been identified, a relatively simplified food web can be constructed that

comprises those species that appear to dominate the biomass at each trophic level of the community. The Site food web is depicted in Figure 4-3. The food web consists of phytoplankton as the primary producers in the River. The primary consumers consist of zooplankton in the water column, and polycheate/oligochaete worms, in the sediments.

The mummichog is the secondary consumer in the food web. The blue crab and striped bass are the tertiary consumers in the food web. The striped bass was chosen as the representative tertiary consumer (over the American eel), because of its importance in the estuarine food web throughout the east coast of the United States, and because of it's commercial and recreational importance to humans. In addition, the early life stages (i.e., eggs and sac-fry larvae) of striped bass are known to be sensitive to the stress of chemical contaminants, particularly chlorinated organic compounds. The selection of striped bass on this basis is consistent with the IWP and EPA guidance (1989, 1992a, 1994a) for selection of representative indicator organisms for risk assessment purposes.

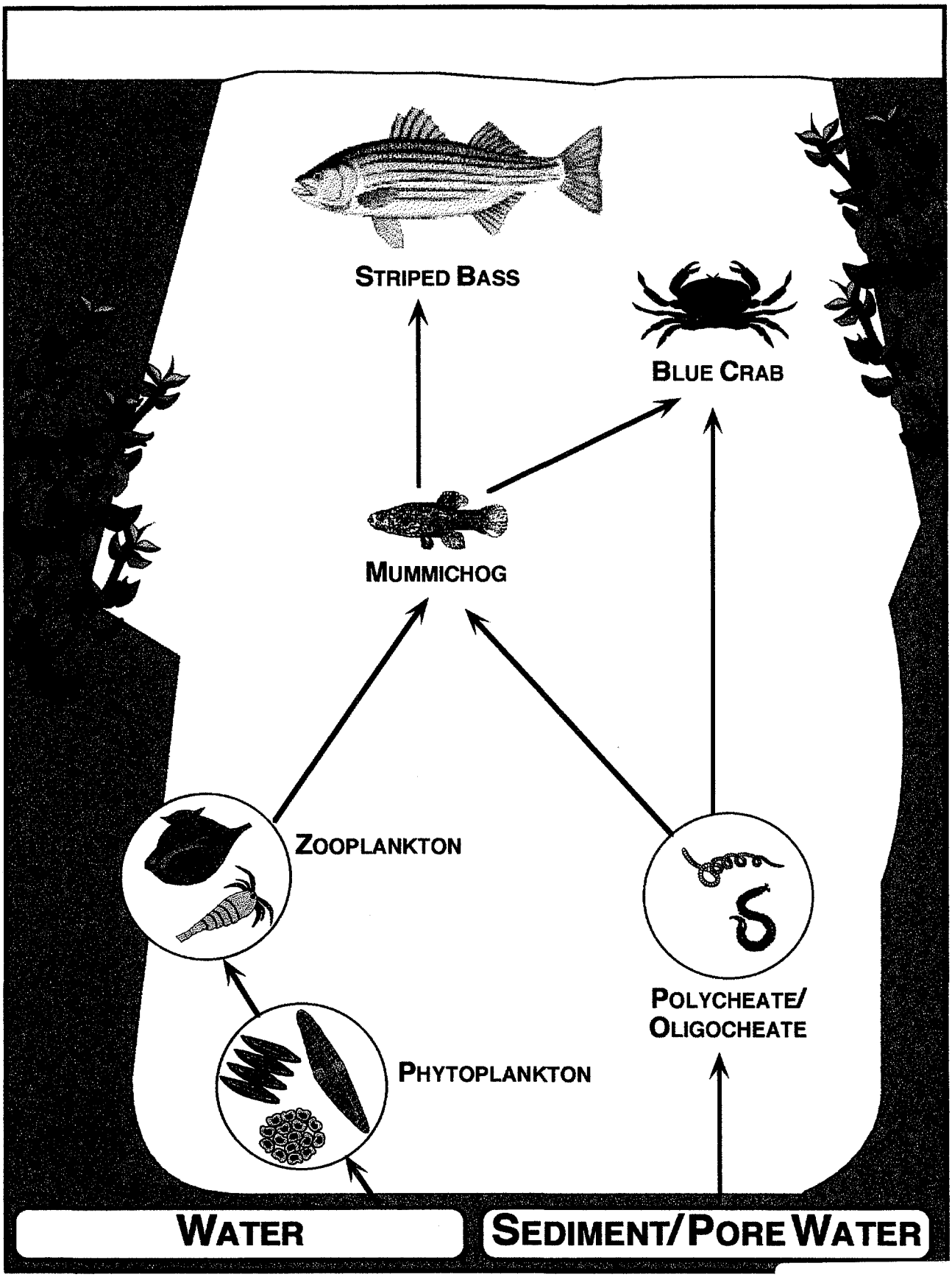
Consistent with the IWP, this simplified food web will serve as the basis for predicting exposures of key organisms to chemicals as a result of uptake from sediment, water, and food sources in Section 4.4. The feeding ecology and bioenergetics of each organisms are discussed in Section 4.4.1.1.

4.3 Selection of Chemicals of Potential Concern for Ecological Risk Assessment

In this section, CPC for the screening-level ERA are selected based on the list of preliminary CPC in Site surface sediments presented in Table 2-4. A CPC screening was not performed for chemicals in surface water, as was intended in the IWP, because of the paucity of water quality data collected from the Site, as discussed in Section 2.0.

As discussed in Section 3.3, guidance on the selection of CPC for risk assessment is presented in the EPA's *Risk Assessment Guidance for Superfund Volume I, Human Health Evaluation Manual - Interim Final* (1989b). For ecological risk assessments, EPA's *Risk Assessment Guidance for Superfund, Volume II Environmental Evaluation Manual - Interim Final* (1989) suggests that additional chemical-specific factors should also be considered when evaluating CPC for ecological

Figure 4-3. Simplified Food Web for the Passaic River Study Area



risk assessments, including: 1) physicochemical properties, 2) bioaccumulation potential, 3) known toxic effects, and 4) exceedance of potential Applicable, Relevant, and Appropriate Requirements (ARARs), including available regulatory criteria.

As described in the IWP, the screening analysis for CPC for the screening-level ERA is based on comparisons of Site surface sediment data to proposed regulatory sediment quality guidelines (SQG). These guidelines already take into account the physicochemical properties of chemicals, as well as some of their known toxic effects. However, the proposed regulatory SQG do not take into account the potential bioaccumulation of chemicals in aquatic organisms, and the consequences of chemical residues in organisms. Therefore, as an additional step in the screening process, a bioaccumulation screening was performed, as previously described in Section 3.3. The primary concern regarding bioaccumulation is that some chemicals that may be present in sediments at low concentrations and, therefore, do not exceed proposed SQG, may nonetheless accumulate to high concentrations in aquatic organisms. The concentrations of such chemicals may be biomagnified within the food web, particularly in higher organisms, such as predatory fish and crabs, that ingest substantial quantities of contaminated prey. The concentrations of chemicals in aquatic organisms may cause adverse effects to the organism, as well as pose substantial risks to predators (including humans) that feed on contaminated prey.

Consistent with EPA guidance (1991), organic chemicals are considered to be bioaccumulative if their log K_{ow} is greater than 3.5. Similar screening values are not available for inorganic chemicals. Therefore, to be conservative, it was assumed that all inorganic chemicals, other than cyanide and essential nutrients, are potentially bioaccumulative. This assumption has been supported by the results of a number of studies on chemical concentrations in fish and other aquatic organisms collected from marine and estuarine environments, including the NY/NJ Harbor Estuary (NJMSC, 1987; NOAA, 1981, 1990, 1995). These studies have demonstrated that most metals can accumulate in aquatic organisms to concentrations substantially above the equilibrium (i.e., background) concentrations that are normally present in an organisms' tissues. As described in the IWP, the following chemicals are considered to be essential nutrients and were not retained for quantitative ecological risk assessment: calcium, iron, magnesium, potassium, and sodium.

The screening analysis for the ERA is presented in Table 4-7. The 95% upper confidence limit (95% UCL) of the arithmetic mean of the Site data for each chemical that was detected in surface sediments was compared to available marine and estuarine SQG. Sediment quality guidelines have been proposed by a number of regulatory agencies, although none have been promulgated to date. These guidelines include NOAA's Effect Range-Median/Effect Range-Low (ER-M/ER-L) values (Long et al., 1995; Long and Morgan, 1991), Washington State's Apparent Effects Threshold (AET) (WADOE, 1991), and values proposed by the Florida Department of Environmental Protection (MacDonald, 1993), as well as EPA's proposed guidelines based on Equilibrium Partitioning (EqP) (EPA, 1993). Preliminary AET values generated (but not currently proposed) by the State of California's Water Resources Control Board (CASWRCB, 1990) were also used to evaluate chemicals for which no other guidelines were available.

Chemicals for which the 95% UCL of the Site surface sediment data exceed the lowest available SQG, and/or those that are potentially bioaccumulative, are considered CPC for the screening-level ERA. Those chemicals that are not potentially bioaccumulative and, for which no available sediment quality guidelines exist, were not retained as CPC. Because there has not been any regulatory attention given to deriving SQG for these chemicals (throughout the U.S), and because they are not considered bioaccumulative, it was assumed that their toxicological significance in sediments is relatively low.

Table 4-8 contains the final list of the CPC for the screening-level ERA. In general, PCDD/Fs and PCBs, as well as most PAHs, pesticides, and inorganic chemicals were retained as CPC. Chemicals that were not retained as CPC were primarily volatile organic and a number of semi-volatile organic compounds that are not considered bioaccumulative, based on the screening analysis described above.

4.4 Exposure Assessment

Consistent with EPA guidance (1989, 1992b, 1994a), the exposure assessment integrates information on the ecological community and CPC, in order to quantify potential exposure of the key organisms to chemicals in the sediments and surface water from the Site. In Section 4.4, potential exposure pathways for key organisms are identified and evaluated through a food web exposure analysis.

Table 4-7. Screening for Chemicals of Potential Concern (CPC) for Ecological Risk Assessment

	Proposed Marine Sediment Quality Guidelines							Summary Statistics for Passaic River Study Area				Screening Evaluation			
	NOAA 1995(b)		WADOE 1991(f)		FDER 1993(j)		Other As					95% UCL Exceeds	No Sediment	Potentially	
	ER-L(c,d)	ER-M(d,e)	SQC(g,h)	MCL(g,i)	NOEL (k)	PEL (l)	Specified (m)	Minimum	Maximum	Mean	95% UCL on the Mean	Minimum Criteria	Criteria Available	Bioaccumuative Chemical (u)	CPC
Inorganics (ppm):															
Aluminum								4,550	24,100	13,100	14,600		x	x	x
Antimony							2 (a,n)	15.6	39.6	7.9	10	x		x	x
Arsenic	8.2	70	57	93	8	64	7.24 (a,q)	3.3	62.3	13	15	x		x	x
Barium								33.7	1,280	179	229		x	x	x
Beryllium								0.3	3.1	1.0	1		x	x	x
Cadmium	1.2	9.6	5.1	6.7	1	7.5	0.67 (a,q)	0.76	14	6.3	7	x		x	x
Chromium	81	370	260	270	33 (a)	240	52.3 (q)	25.8	402	158	179	x		x	x
Cobalt								5.6	41.1	14	15		x	x	x
Copper	34	270	390	390	28	170	18.7 (a,q)	26.4	437	237	260	x		x	x
Lead	46.7	218	450	530	21 (a)	160	30.2 (q)	31.3	840	359	395	x		x	x
Manganese								134	875	383	430		x	x	x
Mercury	0.15	0.71	0.41	0.59	0.1 (a)	1.4	0.13 (q)	0.57	8.1	3.4	4	x		x	x
Nickel	20.9	51.6					15.9 (a,q)	16.8	178	57.3	65	x		x	x
Selenium							0.2 (a,o)	0.78	3.3	1.2	2	x		x	x
Silver	1	3.7	6.1	6.1	0.5 (a)	2.5	0.73 (q)	1.2	39.5	5.3	7	x		x	x
Thallium								0.25	1.9	0.52	1		x	x	x
Titanium								212	605	420	493		x	x	x
Vanadium								18.7	80.6	39.6	43		x	x	x
Zinc	150	410	410	960	68 (a)	300	124 (q)	76.6	1,060	575	628	x		x	x
Cyanide								0.29	269	9.3	24		x		
Organics:															
PCBs (ppb)															
TCB, 3,3',4,4'-								0.018	86	9.0	13		x	x	x
PeCB, 2',3,4,4',5-								0.67	7.1	4.1	5		x	x	x
PeCB, 2,3',4,4',5-								0.13	320	43	57		x	x	x
PeCB, 2,3,3',4,4'-								0.052	190	19	27		x	x	x
PeCB, 2,3,4,4',5-								0.17	2.4	1.3	2		x	x	x
PeCB, 3,3',4,4',5-								0.035	2	0.29	0		x	x	x
HxCB, 2,3',4,4',5,5'-								1.1	14	7.6	9		x	x	x
HxCB, 2,3,3',4,4',5'-								0.18	3.5	1.5	2		x	x	x
HxCB, 2,3,3',4,4',5-								0.65	9.6	4.7	6		x	x	x
HxCB, 3,3',4,4',5,5'-								0.0051	0.078	0.018	0		x	x	x
HpCB, 2,3,3',4,4',5,5'-								0.14	4.3	1.8	2		x	x	x
Aroclor 1248							30 (a,r)	53.5	6,020	548	816	x		x	x
Aroclor 1254							60 (a,r)	485	918	139	201	x		x	x
Total Aroclor PCBs (v)	22.7	180	120	650	24	260	21.5 (a,q)				1,017	x		x	x
Semivolatiles (ppb)															
Bis(2-ethylhexyl)phthalate			470	780			182 (a,q)	960	43,000	15,000	18,000	x		x	x
Butly benzyl phthalate			49 (a)	640				140	920	550	670	x		x	x
Di-n-butyl phthalate			2,200 (a)	17,000				230	820	590	710			x	x
Di-n-octyl phthalate			580 (a)	45,000				110	5,000	680	900	x		x	x
Dichlorobenzene,1,4-			31 (a)	90				130	1,800	590	720	x			x
Dimethyl phthalate			530 (a)	530				1,100	1,100	570	690	x			x
Trichlorobenzene, 1,2,4-			8.1 (a)	18				2,500	2,500	610	750	x		x	x

Table 4-7. Screening for Chemicals of Potential Concern (CPC) for Ecological Risk Assessment

	Proposed Marine Sediment Quality Guidelines							Summary Statistics for Passaic River Study Area				Screening Evaluation			
	NOAA 1995(b)		WADOE 1991(f)		FDER 1993(j)		Other As					95% UCL Exceeds	No Sediment	Potentially	
	ER-L(c,d)	ER-M(d,e)	SQC(g,h)	MCL(g,i)	NOEL (k)	PEL (l)	Specified (m)	Minimum	Maximum	Mean	95% UCL on the Mean	Minimum Criteria	Criteria Available	Bioaccumuative Chemical (u)	CPC
PAHs (ppb)															
Acenaphthene	16	500	160	570	22	450	6.71 (a,q)	230	3,800	710	900	x		x	x
Acenaphthylene	44	640	660	660			5.9 (a,q)	140	1,000	540	660	x		x	x
Anthracene	85.3	1,100	2,200	12,000	85	740	46.9 (a,q)	87	5,100	820	1,100	x		x	x
Benzo(a)anthracene	261	1,600	1,100	2,700	160	1,300	74.8 (a,q)	300	5,800	1,600	1,900	x		x	x
Benzo(a)pyrene	430	1,600	990	2,100	230	1,700	88.8 (a,q)	300	4,300	1,800	2,000	x		x	x
Benzo(b)fluoranthene								310	4,300	1,800	2,000		x	x	x
Benzo(k)fluoranthene							490 (a,p)	200	6,300	1,700	2,000	x		x	x
Benzo(ghi)perylene			310 (a)	780				170	2,500	1,100	1,300	x		x	x
Carbazole								120	1,400	600	720		x		
Chrysene	384	2,800	1,100	4,600	220	1,700	107.8 (a,q)	340	5,900	1,800	2,200	x		x	x
Dibenzo(ah)anthracene	63.4	260	120	330	31	320	6.22 (a,q)	140	1,500	640	760	x		x	x
Dibenzofuran			150 (a)	580				250	3,000	620	780	x		x	x
Fluoranthene	600	5,100	1,600	12,000	380	3,200	113 (a,q)	660	11,000	3,500	4,200	x		x	x
Fluorene	19	540	230	790	18 (a)	460	21.2 (q)	180	4,300	680	880	x		x	x
Indeno(1,2,3-cd)pyrene			340 (a)	880				200	2,500	1,200	1,400	x		x	x
Methylnaphthalene, 2-	70	670	380	640			20.2 (a,q)	160	4,300	660	850	x		x	x
Naphthalene	160	2,100	990	1,700	130	1,100	34.6 (a,q)	550	6,500	790	1,100	x		x	x
Phenanthrene	240	1,500	1,000	4,800	140	1,200	86.7 (a,q)	210	14,000	1,900	2,600	x		x	x
Pyrene	665	2,600	10,000	14,000	290	1,900	153 (a,q)	630	11,000	3,200	3,900	x		x	x
L-PAHs(s)	552	3,160	3,700	7,800	250 (a)	2,400		87	14,000	870	1,000	x			x
H-PAHs(t)	1,700	9,600	9,600	53,000	870 (a)	8,500		120	11,000	1,600	1,800	x			x
Total PAHs	4,022	44,792			2,900 (a)	28,000	4,000 (n)				34,050	x			x
Pesticides (ppb)															
Aldrin							5 (a,r)	4.81	59.8	7.7	11	x		x	x
beta-BHC							2 (a,r)	3.14	56.2	4.46	7	x		x	x
Delta-BHC							5 (a,r)	4.67	23.8	4.42	6				
Chlordane							1 (a,n)	18.0	18.0	18.0	NA (w)	x		x	x
alpha-Chlordane								3.5	66	17.0	21		x	x	x
gamma-Chlordane								3.39	117	18.8	24		x	x	x
DDD, 4,4'-							1.22 (a,q)	5.59	591	109	150	x		x	x
DDE, 4,4'-	2.2	27			1.7 (a)	130	2 (n)	11.5	106	42.7	50	x		x	x
DDT, 4,4'-							1 (a,n)	6.19	293	37	53	x		x	x
Dieldrin							0.72 (a,q)	7.93	270	17	28	x		x	x
Endrin								19	134	19.8	27			x	x
Methoxychlor								32.7	445	35	55		x	x	x
Misc. Organics (ppb)															
TEPH (ppm)								30	2,740	875	1,120		x	(x)	(x)
Dibutyltin								742	742	193	335		x	x (y)	x
Monobutyltin								276	835	328	471		x	x (y)	x
PCDD/Fs (ppt)															
TCDD, 2,3,7,8-								2	1,600	340	420		x	x	x
PECDD, 1,2,3,7,8-								2.3	47	9.4	11		x	x	x
HxCDD, 1,2,3,4,7,8-								0.92	93	10	14		x	x	x
HxCDD, 1,2,3,6,7,8-								2.7	120	37	44		x	x	x
HxCDD, 1,2,3,7,8,9-								1.5	53	18	21		x	x	x
HpCDD, 1,2,3,4,6,7,8-								5.6	2,070	570	680		x	x	x
OCDD								135	81,000	7,500	11,000		x	x	x
Total TCDD								2	1,700	460	560		x	x	x
Total PECDD								4.4	1,190	100	150		x	x	x
Total HXCDD								7	1,100	320	390		x	x	x

Table 4-7. Screening for Chemicals of Potential Concern (CPC) for Ecological Risk Assessment

	Proposed Marine Sediment Quality Guidelines							Summary Statistics for Passaic River Study Area				Screening Evaluation			
	NOAA 1995(b)		WADOE 1991(f)		FDER 1993(j)		Other As					95% UCL Exceeds	No Sediment	Potentially	
	ER-L(c,d)	ER-M(d,e)	SQC(g,h)	MCL(g,i)	NOEL (k)	PEL (l)	Specified (m)	Minimum	Maximum	Mean	95% UCL on the Mean	Minimum Criteria	Criteria Available	Bioaccumuative Chemical (u)	CPC
Total HPCDD								20	5,890	1,300	1,500		x	x	x
TCDF, 2,3,7,8-								1.8	280	39	52		x	x	x
PECDF, 1,2,3,7,8-								1.5	580	28	50		x	x	x
PECDF, 2,3,4,7,8-								4	1,400	80	130		x	x	x
HxCDF, 1,2,3,4,7,8-								8.6	20,000	610	1,400		x	x	x
HxCDF, 1,2,3,6,7,8-								2.6	2,900	110	220		x	x	x
HxCDF, 1,2,3,7,8,9-								0.54	300	14	25		x	x	x
HxCDF, 2,3,4,6,7,8-								2.8	780	48	77		x	x	x
HpCDF, 1,2,3,4,6,7,8-								2.6	64,000	2,100	4,500		x	x	x
HpCDF, 1,2,3,4,7,8,9-								1.1	1,400	53	110		x	x	x
OCDF								50	130,000	3,800	8,700		x	x	x
Total TCDF								4.3	6,700	770	1,000		x	x	x
Total PECDF								4.8	11,000	850	1,300		x	x	x
Total HXCDF								5.6	36,000	1,500	2,900		x	x	x
Total HPCDF								2.6	76,000	2,800	5,700		x	x	x

a. Minimum reported screening guidelines for a chemical
b. National Oceanic Atmospheric Administration (NOAA) values for marine and estuarine sediments reported in Long et al. (1995)
c. Effect range-low
d. Values reported in dry weight
e. Effect range-median
f. Washington State Department of Ecology
g. Organic values normalized to 1 percent organic carbon for Passaic River sediments; inorganic values reported on a dry weight basis
h. Sediment Quality Criteria
i. Minimum cleanup levels developed for Puget Sound
j. Florida Department of Environmental Regulation values for marine and estuarine sediments reported in MacDonald et al. (1993)
k. No Observed Effect Level
l. Permissible Effect Level
m. Where more than one other value was available, the minimum reported guideline was selected for consideration
n. ER-L value as reported in Long and Morgan (1990)
o. Amphipod Apparent Effects Threshold (AET) reported in CASWRCB, 1990
p. Benthic Apparent Effects Threshold (AET) reported in CASWRCB, 1990
q. Environment Canada (1994) Threshold Effect Levels (TEL)
r. Ontario Ministry of the Environment Lowest Effect Levels (LEL) (Persaud, 1993)
s. Low-molecular-weight PAHs
t. High-molecular-weight PAHs
u. Organic chemicals with a log Kow > 3.5 were considered to be potentially bioaccumulative (EPA, 1991); All metals were considered to be potentially bioaccumulative
v. The concentration of Total PCBs is defined as the sum of the individual Aroclor mixtures
w. 95% UCL could not be calculated, therefore the maximum value was used for comparison to Sediment Quality Guidelines
x. Total extractable petroleum hydrocarbons (TEPH) are considered to be potentially bioaccumulative CPC, and are evaluated in the screening-level ERA based on PAH constituents
y. Organotins are potentially bioaccumulative, similar to other metals

Table 4-8. Chemicals of Potential Concern (CPC) for Ecological Risk Assessment

Inorganics	PCBs	Semivolatiles	PAHs	Pesticides	Misc. Organics	PCDD/Fs
Aluminum	TCB, 3,3',4,4'-	Bis(2-ethylhexyl)phthalate	Acenaphthene	Aldrin	Dibutyltin	TCDD, 2,3,7,8-
Antimony	PeCB, 2',3,4,4',5-	Butly benzyl phthalate	Acenaphthylene	Beta-BHC	Monobutlytin	PECDD, 1,2,3,7,8-
Arsenic	PeCB, 2,3',4,4',5-	Di-n-butyl phthalate	Anthracene	Chlordane		HxCDD, 1,2,3,4,7,8-
Barium	PeCB, 2,3,3',4,4'-	Di-n-octyl phthalate	Benzo(a)anthracene	alpha-Chlordane		HxCDD, 1,2,3,6,7,8-
Beryllium	PeCB, 2,3,4,4',5-	Dimethyl phthalate	Benzo(a)pyrene	gamma-Chlordane		HxCDD, 1,2,3,7,8,9-
Cadmium	PeCB, 3,3',4,4',5-	Trichlorobenzene, 1,2,4-	Benzo(b)fluoranthene	Dieldrin		HpCDD, 1,2,3,4,6,7,8-
Chromium	HxCB, 2,3',4,4',5,5'-		Benzo(k)fluoranthene	DDD, 4,4'-		OCDD
Cobalt	HxCB, 2,3,3',4,4',5'-		Benzo(g,h,i)perylene	DDE, 4,4'-		Total TCDD
Copper	HxCB, 2,3,3',4,4',5-		Chrysene	DDT, 4,4'-		Total PECDD
Lead	HxCB, 3,3',4,4',5,5'-		Dibenzo(ah)anthracene	Endrin		Total HXCDD
Manganese	HpCB, 2,3,3',4,4',5,5'-		Dibenzofuran	Methoxychlor		Total HPCDD
Mercury	Aroclor 1248		Fluoranthene			TCDF, 2,3,7,8-
Nickel	Aroclor 1254		Fluorene			PECDF, 1,2,3,7,8-
Selenium	Total PCB		Indeno(1,2,3-cd)pyrene			PECDF, 2,3,4,7,8-
Silver			Methylnaphthalene, 2-			HxCDF, 1,2,3,4,7,8-
Thallium			Naphthalene			HxCDF, 1,2,3,6,7,8-
Titanium			Phenanthrene			HxCDF, 1,2,3,7,8,9-
Vanadium			Pyrene			HxCDF, 2,3,4,6,7,8-
Zinc			Low Molecular Weight PAHs			HpCDF, 1,2,3,4,6,7,8-
			High Molecular Weight PAHs			HpCDF, 1,2,3,4,7,8,9-
			Total PAHs			OCDF
						Total TCDF
						Total PECDF
						Total HXCDF
						Total HPCDF

4.4.1 Identification of Potential Exposure Pathways for Key Organisms

Consistent with EPA guidance (EPA, 1989, 1992b, 1994a), exposure pathways for key organisms were evaluated. A complete ecological exposure pathway should include the following elements:

- a source and mechanism of chemical release to the environment
- an environmental transport medium (e.g., water, sediment, biota)
- an ecological exposure route at the contact point (e.g., ingestion, dermal contact)

The sources and mechanisms of chemical release to the Site were previously discussed in the Section 4.1. For each key organism identified, potential exposure to chemicals in surface water, biota, and sediments are considered. Potential pathways of ecological exposure to chemicals in these media include:

Surface Water

- ingestion/uptake of surface water;

Sediment

- ingestion/uptake of sediment;
- direct contact with sediment; and,

Biota

- consumption of aquatic organisms.

For all aquatic organisms, uptake of chemicals can occur from exposure to contaminated water, sediment, and food sources. The contributions of chemicals from each of these media vary between species, and are dependent on the life history, particularly the feeding ecology, of an organism, as well as the physicochemical properties of the chemical. The exposure pathways for key organisms at each trophic level that were identified in Section 4.2.5.1, are discussed below.

4.4.1.1 Plankton

The uptake of chemicals by marine and estuarine phytoplankton and zooplankton occurs via respiration across the body integument and, for zooplankton, from assimilation of chemicals from

contaminated food sources (Clayton et al., 1977; Wyman and O'Connors, 1980). The large surface-to-volume ratio of these organisms likely contributes to the efficient uptake of chemicals via respiration from water, unlike larger organisms at higher trophic levels which accumulate chemicals primarily from ingestion of food and sediments (Clayton et al., 1977; Thomann et al. 1992; Gobas 1993). For this reason, the absorption of chemicals from water is believed to be the most important exposure pathway in plankton (Clayton et al., 1977; Gobas 1993). Therefore, for the purposes of the screening-level ERA, it was assumed that absorption of chemicals from the water column across the cell membrane is the only exposure route for planktonic communities.

4.4.1.2 Polychaetes/Oligochaetes

Polychaetes and oligochaetes are borrowing annelid worms (i.e., infauna) that live in sediments. Most are detritivores, consuming sediment, detritus, and to a lesser extent, plankton (Kay and Brafield, 1972). Uptake of chemicals by polychaetes and oligochaetes may occur via respiration of interstitial water, ingestion of sediments/sediment-associated detritus, and absorption from sediments (Rubinstein et al., 1983). However, several investigators have demonstrated that chemicals are accumulated by polychaetes and oligochaetes primarily from sediments (Courtney and Langston, 1978; Fowler et al., 1978; McLeese et al., 1980; Oliver, 1984; Pruell et al., 1993). Thus, ingestion of and absorption from sediments is considered to be the primary exposure pathway for accumulation of chemicals in polychaetes and oligochaetes at the Site.

4.4.1.3 Mummichog

Mummichogs are secondary consumers (i.e., forage fish) that feed primarily on benthic invertebrates, plankton, and sediment-associated detritus (Valiela et al., 1977; Kneib and Stiven, 1978; Weisberg et al., 1981; USFWS, 1985). Forage fish obtain the vast majority of their chemical intake through ingestion of contaminated food sources (Connolly, 1991; Thomman et al., 1992; Gobas, 1993). In this screening-level assessment, it was assumed that mummichogs at the Site are feeding on polychaetes/oligochaetes and plankton at a ratio of 1:1. At the Site, it is likely that mummichog are the primary food source for both blue crab and striped bass, as well as other less abundant predatory organisms.

4.4.1.4 Blue Crab

As illustrated in Figure 4-3, blue crabs occupy a mid-trophic level at the Site and are primarily tertiary consumers. Predatory species obtain the vast majority of their chemical intake through ingestion of contaminated food sources (Connolly, 1991; Thomman et al., 1992; Gobas, 1993). Blue crabs are scavengers that feed on a number of available food sources. Given the variability in the reported food sources of blue crabs in various east coast estuaries, it appears that the blue crab's diet is depends primarily on the food that is available. The primary food sources for blue crabs in east coast estuaries are fish, shellfish, benthic invertebrates, and detritus (Kneib and Stiven, 1982; Laughlin 1982; West and Williams, 1986; USFWS, 1989). Based on the limited prey that are available for blue crabs at the Site, it is likely that their primary food sources are mummichogs and polychaetes/oligochaetes. For this analysis, it was assumed that blue crabs at the Site feed on mummichogs and polychaetes/oligochaetes at the Site at a ratio of 3:1, respectively.

4.4.1.5 Striped Bass

As illustrated in Figure 4-3, striped bass occupy the highest trophic level at the Site and are primarily tertiary consumers. Predatory fish obtain the vast majority of their chemical intake through ingestion of contaminated food sources (Connolly, 1991; Thomman et al., 1992; Gobas, 1993). A number of studies have been conducted to determine the feeding preferences of striped bass (Setzler et al., 1980). These studies suggest that the diet of striped bass is strongly dependant on the size and age of the individual. Specifically, adult striped bass have been reported to be primarily piscivorous, while juveniles consume a significant proportion of water column and benthic invertebrates. Manooch (1973) evaluated the food habits of adult striped bass from Albemarle Sound, NC and reported that fish, particularly small clupeids (alewife, gizzard shad, and Atlantic menhaden), occurred in 93% of the stomachs that contained undigested or partially digested food. Similarly, Manooch (1973) found that the primary source of food consumed by adult striped bass from Long Island Sound consisted of fish species, particularly bay anchovy. The results of these investigations suggest that the fish species ingested by adult striped bass are largely dependent on prey size and availability. For this reason, it is most likely that striped bass that forage in the tidal Passaic River are feeding primarily on mummichog, which are the most abundant fish species at the Site. Thus, for this analysis, it was assumed that striped bass feed

entirely on mummichogs at the Site. This assumption is conservative and will have a significant impact on the exposure estimates for striped bass, since mummichogs (at the Site) are confined to a small home range and, therefore, are exposed to chemicals in sediments and food at the Site throughout the year. Other species, such as clupeids or bay anchovy, that may comprise a portion of the striped bass diet at the Site, are not year-round residents in the tidal Passaic River.

4.4.2 Food Web Exposure Analysis

A screening-level exposure analysis was performed to estimate the potential accumulation of organic and inorganic CPC in key organisms at the Site. Consistent with EPA guidance (1992a, 1994a) on conducting screening-level evaluations, conservative assumptions were used in the absence of Site-specific data.

4.4.2.1 Exposure Point Concentrations

Recent data regarding concentrations of chemicals in fish and other aquatic organisms from the Site are not available. Available data for organic chemicals in fish and blue crab that were collected in the mid-1980s are summarized in Table 2-2. Because of the high sediment accretion rates at the Site (average = about 1.7 to 2.6 cm/yr; see Section 2.0), and subsequent declines in chemical concentrations in sediments (and presumably fish and crabs) over the last ten years, these data are not considered reflective of current conditions, and were not used to evaluate the present risks that CPC may pose to aquatic organisms. There have not been data collected from the Site regarding concentrations of inorganic chemicals and many organic chemicals in key organisms.

To estimate current concentrations of organic chemicals in key organisms, a screening-level food web exposure analysis was conducted. The analysis considers exposures of key organisms at the Site to chemicals in sediments, surface water, and food sources (i.e., prey). The analysis was conducted using conservative exposure assumptions for the key organisms at the Site, and was based on the highest exposure point concentrations (i.e., 95% UCL of the arithmetic mean) found in surface sediments from the Site. Because surface water data are not available for the Site, surface water concentrations of organic chemicals were approximated in the model using the relationship $C_{sw} = C_{ss}/K_{ow}$, where C_{sw} is the estimated concentration of chemical in surface water, and C_{ss} is the 95% UCL of the arithmetic mean of the Site surface sediment data. This is likely an

overly conservative approximation, since it does not take into account the dissolved or particulate organic carbon content of either sediment or water, or other factors that severely limit the concentrations of organic chemicals in surface water. However, for the purposes of the screening-level ERA, this relationship was assumed to represent the chemicals available for uptake via water. The K_{ow} for organic CPC are reported in Table 4-9.

For inorganic chemicals, it is not currently possible to estimate chemical concentrations in aquatic organisms from concentrations in sediments using a mechanistic model. In addition, there are no empirical bioaccumulation factors (BAFs) published for inorganic chemicals. There are several reasons for this, most notably because of the large number of physicochemical factors associated with chemical complexation in sediments and organisms, and the substantial variation of metal sequestration in various organisms or phylogenetic groups make the modeling of such factors a very complex exercise. To that end, only empirical estimates of bioaccumulation of inorganic chemicals based on sediment data can be made by evaluating the limited data from the scientific literature that presents concurrent measurements of chemical concentrations in sediments and aquatic organisms. For this analysis, the limited data from the NY/NJ Harbor Estuary was evaluated to select a conservative partition coefficient for metals to estimate potential concentrations that may be expected in key organisms at the Site, based on the inorganic chemical concentrations in surface sediments. The inorganic analysis and results are presented in Section 4.4.2.4.

4.4.2.2 Description of Food Web Model

A food web model with sediment interaction was used to estimate steady-state whole body concentrations of organic CPC in key organisms. The model was constructed for the simplified food web depicted in Figure 4-3 and is based on surface sediment and water concentrations of CPC, as well as a number of bioenergetics-based exposure parameters for the key organisms at the Site.

A modified version of the food web model that was presented in the IWP (i.e., Thomann et al., 1992) was used in this analysis. A number of parameters in the model were modified to conform to recent improvements in food web modeling described by Gobas (1993). These improvements generally simplify the assumptions used to estimate trophic transfer of chemicals in a food web by taking advantage of a number of well-documented thermodynamic relationships between the

Table 4-9. Chemical-Specific Kow Values for CPC

CPC	Kow	log Kow	Reference
PAHs			
Acenaphthene	9,600	3.98	EPA, 1982
Acenaphthylene	5,300	3.72	EPA, 1982
Anthracene	28,000	4.45	EPA, 1982
Benzo(a)anthracene	410,000	5.61	EPA, 1982
Benzo(a)pyrene	$1.15x10^6$	6.06	EPA, 1982
Benzo(b)fluoranthene	$1.15x10^6$	6.06	EPA, 1982
Benzo(ghi)perylene	$3.20x10^6$	6.51	EPA, 1982
Benzo(k)fluoranthene	$1.15x10^6$	6.06	EPA, 1982
Chrysene	410,000	5.61	EPA, 1982
Dibenzo(a,h)anthracene	$6.90x10^6$	6.84	EPA, 1982
Dibenzofuran	13,200	4.12	HSDB, 1995
Fluoranthene	79,000	4.90	EPA, 1982
Fluorene	15,000	4.18	EPA, 1982
Indeno(1,2,3-c,d)pyrene	$3.20x10^6$	6.51	EPA, 1982
Methylnaphthalene, 2-	7,240	3.86	HSDB, 1995
Phenanthrene	28,000	4.45	EPA, 1982
Pyrene	80,000	4.90	EPA, 1982
Pesticides			
Aldrin	200,000	5.30	EPA, 1982
Alpha-Chlordane	300,000	5.48	EPA, 1982
Beta-BHC	7,800	3.89	EPA, 1982
DDD, 4,4'-	$1.60x10^6$	6.02	HSDB, 1995
DDE, 4,4'-	$9.10x10^6$	6.51	HSDB, 1995
DDT, 4,4'-	$8.10x10^6$	6.36	HSDB, 1995
Dieldrin	3,500	3.54	EPA, 1982
Endrin	398,000	5.60	HSDB, 1995
Gamma-Chlordane	300,000	5.48	EPA, 1982
Methoxychlor	67,600	4.83	HSDB, 1995
PCBs			
IUPAC #189, 2,3,3',4,4',5,5'-HeptaCB	$5.13x10^7$	7.71	EPA, 1994a
IUPAC #156, 2,3,3',4,4',5-HexaCB	$1.51x10^7$	7.18	EPA, 1994a
IUPAC #157, 2,3,3',4,4',5'-HexaCB	$1.58x10^7$	7.20	EPA, 1995b
IUPAC #167, 2,3',4,4',5,5'-HexaCB	$2.00x10^7$	7.30	EPA, 1995b
IUPAC #169, 3,3',4,4',5,5'-HexaCB	$2.95x10^7$	7.47	EPA, 1995b
IUPAC #118, 2,3',4,4',5-PentaCB	$5.50x10^6$	6.74	EPA, 1994a
IUPAC #126, 3,3',4,4',5-PentaCB	$7.76x10^6$	6.89	EPA, 1994a
IUPAC #105, 2,3,3',4,4'-PentaCB	$4.47x10^6$	6.65	EPA, 1994a
IUPAC #114, 2,3,4,4',5-PentaCB	$3.98x10^6$	6.60	EPA, 1995b
IUPAC #123, 2',3,4,4',5-PentaCB	$3.98x10^6$	6.60	EPA, 1995b
IUPAC #77, 3,3',4,4'-TetraCB	$2.29x10^6$	6.36	EPA, 1994a
Aroclor 1248	575,000	5.76	EPA, 1982
Aroclor 1254	$1.10x10^6$	6.04	EPA, 1982

Table 4-9. Chemical-Specific Kow Values for CPC

CPC	Kow	log Kow	Reference
Semivolatiles			
bis(2-Ethylhexyl)phthalate	4.10×10^{9}	4.89	HSBD, 1995
Butyl benzyl phthalate	360,000	5.56	EPA, 1982
Di-n-butyl phthalate	360,000	5.56	EPA, 1982
Di-n-octyl phthalate	7.40×10^{9}	5.22	HSBD, 1995
Trichlorobenzene, 1,2,4-	19,000	4.28	EPA, 1992
PCDD/Fs			
TCDD, 2,3,7,8-	1.05×10^{7}	7.02	EPA, 1995b
PECDD, 1,2,3,7,8-	3.16×10^{7}	7.50	EPA, 1995b
HxCDD, 1,2,3,4,7,8-	6.31×10^{7}	7.80	EPA, 1995b
HxCDD, 1,2,3,6,7,8-	6.31×10^{7}	7.80	EPA, 1995b
HxCDD, 1,2,3,7,8,9-	6.31×10^{7}	7.80	EPA, 1995b
HpCDD, 1,2,3,4,6,7,8-	1.58×10^{8}	8.20	EPA, 1995b
OCDD	3.98×10^{8}	8.60	EPA, 1995b
TCDF, 2,3,7,8-	631,000	5.80	EPA, 1995b
PECDF, 1,2,3,7,8-	3.16×10^{6}	6.50	EPA, 1995b
PECDF, 2,3,4,7,8-	1.00×10^{7}	7.00	EPA, 1995b
HxCDF, 1,2,3,4,7,8-	3.16×10^{7}	7.50	EPA, 1995b
HxCDF, 1,2,3,6,7,8-	3.16×10^{7}	7.50	EPA, 1995b
HxCDF, 1,2,3,7,8,9-	3.16×10^{7}	7.50	EPA, 1995b
HxCDF, 2,3,4,6,7,8-	3.16×10^{7}	7.50	EPA, 1995b
HpCDF, 1,2,3,4,6,7,8-	1.00×10^{8}	8.00	EPA, 1995b
HpCDF, 1,2,3,4,7,8,9-	1.00×10^{8}	8.00	EPA, 1995b
OCDF	6.31×10^{8}	8.80	EPA, 1995b

physicochemical properties of organic compounds and their biological activity (particularly bioavailability). The model was run using Microsoft® Excel Version 4.0 for Windows, on a Gateway® PC486 computer. The individual model spreadsheets, depicting the input parameters, calculations, and results are provided as Appendix G.

The accumulation of nonionic organic chemicals from sediments, water, and a variety of food sources can be described for multiple organisms in a food web by multi-compartment models that adequately represent the bioenergetics and feeding interactions of each organism within the food web (Norstrom et al., 1976; Connolly and Tonelli, 1985; Connolly and Pedersen, 1988; Gobas et al., 1988; Thomann, 1989; Connolly, 1991; Fordam and Reagan, 1991; Thomann et al., 1992; Gobas, 1993). Similar to the metabolic pathways for consumption of food, xenobiotic chemicals that are ingested or absorbed by aquatic organisms are either incorporated into body tissues (bioaccumulated), metabolized and/or transformed, or excreted. These pathways can be evaluated using a series of steady state, mass balance equations based on the first law of thermodynamics regarding the conservation of mass and energy (Brett and Groves, 1979; Brandt and Hartman, 1993).

An aquatic organisms' body burden (e.g., whole body concentration) of a chemical can be characterized by the following steady state equation:

$$C_b = U_{water} + U_{sediment} + U_{food} \text{ - Metabolic Loss}$$

where C_b is the body burden of a given chemical in an organism, U_{water} is the direct uptake of a chemical from both sediment interstitial (pore) water and the water column, $U_{sediment}$ is the uptake from ingestion of sediment, U_{food} is the indirect uptake of a chemical from feeding on contaminated organisms, and Metabolic Loss is the direct loss of a chemical from excretion, metabolism, and dilution from growth.

The screening-level model equation and (bioenergetic) exposure parameters that control the uptake of a chemical by an organism under steady state conditions are defined as follows:

$$dC_i/dt = 0 = [k1C_w] + [(p_{ix}\,CAE\,I_{ix})C_x] - [(k2 + kG_i + kM + kE)C_i] \quad (1)$$

or

$$C_i = \{[k1C_w] + [(p_{ix}\,CAE\,I_{ix})C_x]\} / [k2 + kG_i + kM + kE] \quad (2)$$

where:

C_i	=	estimated lipid normalized concentration (µg/kg(lipid)) in predator i
k1	=	rate of chemical uptake from surface waters (L/day-g(lipid))
C_{sw}	=	concentration of chemical in surface water (µg/l)
x	=	prey organism
p_{ix}	=	feeding preference for predator i on prey organism x
CAE	=	chemical assimilation efficiency (g(chemical)-assimilated/g(chemical)-ingested)
I_{ix}	=	ingestion/consumption rate of predator i on prey x (g_x/g_i day)
C_x	=	estimated lipid normalized concentration in prey x (µg/g(lipid))
C_{ss}	=	organic carbon normalized sediment concentration (µg/kg-oc)
k2	=	depuration rate (1/day)
kM	=	rate of chemical metabolism (1/day)
kE	=	excretion rate (1/day)
kG_i	=	growth rate (1/day)

The first bracketed term on the right side of equation 2 $[k1C_w]$ represents the direct uptake of dissolved chemical from the water column. This pathway is generally not significant for highly hydrophobic chemicals because of their extremely low water solubilities.

The second bracketed term $[(p_{ix}CAE_i\ I_{ix})C_x]$ represents the uptake of chemical due to ingestion of prey. The uptake from food is determined by the feeding preference(s) (p) of an organism, its consumption rate (I), and its chemical assimilation efficiency (CAE), which is the fraction or percent of the total amount of a chemical that is ingested from food or sediments or absorbed from water that is accumulated in the body of an organism. The food uptake term is calculated for any number of prey organisms (x) of the predator (i). The third bracketed term $[k2 + kG_i + kM + kE]$ represents the loss of chemical due to depuration (k2), dilution from growth (kG), metabolism (kM), and excretion (kE).

The model was used to estimate chemical accumulation in plankton via water uptake only, and mummichog, blue crab, and striped bass, via food ingestion and, to a lesser extent, water uptake. Direct chemical accumulation from sediments was only considered a pathway for infaunal polychaetes/oligochaetes. For these organisms, biota sediment accumulation factors (BSAF) were incorporated in to the model to estimate chemical accumulation via sediment ingestion/absorption. The BSAF was incorporated as the CAE in the model (for polychaetes/oligochaetes), and the resulting estimated C_i were equal to the product of the organic carbon-normalized surface sediment concentration (C_{ss}/f_{oc}) and the CAE. The BSAFs for polychaetes/oligochaetes were derived from bioaccumulation data collected in the tidal Passaic River by Rubinstein et al. (1990) and Pruell et al. (1993), and other data reported in the scientific literature. The BSAFs were used in the model to avoid the large uncertainties surrounding the bioenergetics of polychaetes and, are appropriate, since infaunal organisms accumulate chemicals exclusively from sediments and pore water. Consistent with the equilibrium partitioning theory (EPA, 1993), BSAFs were assumed to approximate unity (1.0) for most organic CPC including Aroclor PCBs, penta-, hexa-, and hepta-coplanar PCBs, pesticides, and semivolatiles (i.e., PAHs and phthalates). A BSAF of 0.5 was used for 3,3',4,4'-tetraCB and PCDD/Fs, based on Site-specific values reported by Pruell et al. (1993) for these chemicals.

The bioenergetics exposure parameters for fish and blue crab that were used in the screening-level model are presented in Table 4-10. Exposure parameters were derived from available literature regarding the life histories and bioenergetics of the key organisms identified at the Site in Section 4.1.5. For those parameters that are not generally reported in the literature (i.e, excretion rates, respiration rates, and metabolic rates), values were defined by either allometric (body weight) relationships described by Thomann et al. (1992), or as relationships reported by Thomann

Table 4-10. Bioenergenic Exposure Parameters for Key Organisms at the Site (a)

Parameter	Units	Phytoplankton	Zooplankton	Mummichog	Blue Crab	Striped Bass	Polychaete/ Oligochaete	Sources
Organism Weight (W)	g (wet)	0.0001	0.001	3	200 (d)	3000 (e)	NA	Hauge et al., 1990; Belton et al., 1985; Zabik et al., 1991; Hauge et al., 1993; Iannuzzi, 1991; Bush et al., 1988
Fraction Lipid (fL)	gL/g(wet)	0.01	0.05	0.025	0.028 (d)	0.05 (e)	0.01	Hauge et al., 1990; Hauge et al., 1993; Pruell et al., 1993; Kay and Brafield,1972; Belton et al., 1982; Ebasco, 1993; Bush et al., 1988
Food Assimilation Efficiency (FAE)	unitless	NA	NA	0.8	0.45	0.8	NA	Targett, 1979
Wet to Dry Ratio (W/D)	unitless	10	10	5	5	5	7	Kay and Brafield, 1972
Growth Rate (kG) (a)	1/day	NA	NA	0.008	0.003	0.002	0.50	Thomann et al., 1992
Respiration Rate (R) (b)	1/day	NA	NA	0.029	0.012	0.007	NA	Thomann et al., 1992
Oxygen Respiration Rate (r) (c)	gO2/g-day	NA	NA	0.007	0.003	0.002	NA	Parkerton, 1991

NA - Not Applicable

(a) Calculations for other variables, including water uptake (k1), metabolism (kM), chemical assimilation efficiency (CAE), and ingestion rate (I) are discussed in the text on an organism- and/or chemical-specific basis; depuration (k2) and excretion (kE) are calculated in the food web model on a chemcial-specific basis as follows:

k2 = k1/Kow*fL

kE = 0.25*I

(b) kG = 0.01*W^-0.2

(c) R = 0.036*W^-0.2

(d) r = 1.2*R*(1/W/D)

(e) Average wet weight and whole-body lipid content reported for adult striped bass from Hudson River stock

(f) Average wet weight and whole-body lipid content reported for adult blue crab collected from the NY/NJ Harbor Estuary

(1989), Connolly (1991), or Gobas (1993). The pathways for accumulation of chemicals by key organisms, and the primary factors that regulate chemical accumulation from each pathway are discussed below.

Chemical Assimilation Efficiency (CAE)

The CAE of xenobiotic chemicals by estuarine organisms is influenced by the complex feeding interactions within the food web, species-specific bioenergetics, exposure(s) of each organism to contaminated media, physicochemical properties of the water and sediments, and the fate and transport of the chemicals in the estuarine environment (Norstrom et al., 1976; Brett and Groves, 1979; Connolly and Tonelli, 1985; Connolly, 1991; EPA, 1993). Consequently, this parameter may vary between different estuaries or sites when there are significant difference in the aforementioned factors. Because of the site-specificity of this parameter, it is not usually determined *a priori*; rather, it is determined through model calibration using site-specific chemical data for sediments and biota. Alternatively, the CAE may be estimated using empirical relationships established from evaluation of historical accumulation data for various chemical groups and organisms (Thomann, 1989; Thomann et al., 1992; Gobas, 1993). For this screening-level assessment, the relationships between log K_{ow} and CAE described by Thomann (1989) were used to estimate the CAE for all organic bioaccumulative CPC. For mummichogs which have an average wet weight of less than 100 grams, the relationships are:

$$\log CAE = -2.6 + 0.5 \log K_{ow} \quad \text{for } \log K_{ow} = 2 - 5 \tag{3a}$$

$$\log CAE = 0.8 \quad \text{for } \log K_{ow} = 5 - 6 \tag{3b}$$

$$\log CAE = 2.9 - 0.5 \log K_{ow} \quad \text{for } \log K_{ow} = 6 - 9 \tag{3c}.$$

For blue crab and striped bass (adults) which have average wet weights of greater than 100 grams,the relationships are:

$$\log CAE = -1.5 + 0.4 \log K_{ow} \quad \text{for } \log K_{ow} = 2 - 3 \quad (4a)$$

$$\log CAE = 0.5 \quad \text{for } \log K_{ow} = 3 - 6 \quad (4b)$$

$$\log CAE = 1.2 - 0.25 \log K_{ow} \quad \text{for } \log K_{ow} = 6 - 9 \quad (4c).$$

Chemical Accumulation from Water (k1)

For phytoplankton and zooplankton, which accumulate chemicals primarily via respiration across the body surface, k1 is defined as the bioconcentration factor (BCF). The plankton BCF has been described by several investigators (i.e., Thomann, 1989; Connolly, 1991; Gobas, 1993) to approximate $K_{ow}*f_L$, where f_L is the fraction of lipid in plankton. For fish and blue crab, k1 is defined as the chemical accumulated from the water column via respiration across the surface of the gills in L/day-g(wet). The uptake from water across the gills can be related to the oxygen respiration rate of an organism as follows:

$$k1 = r\, C_{ox}(E_c/E_{ox}) \text{ where,} \quad (5a)$$

r = oxygen respiration rate (gO_2/g-day);
C_{ox} = mean concentration of dissolved oxygen in the water column (g/l); and,
E_c/E_{ox} = chemical to oxygen assimilation ratio, and

$$\log E_c/E_{ox} = 3.082 - 0.529 \log K_{ow} \quad (5b).$$

Equation 5b was developed by Parkerton (1991), and describes the efficiency of chemical uptake from water relative to dissolved oxygen uptake for organic chemicals.

Dietary Accumulation of Chemicals

As described by Thomann et al. (1992), dietary accumulation of chemicals by fish and blue crab is a function of their feeding preference(s) at a site, consumption (or ingestion) rates, the chemical concentration in prey organisms, and the CAE for various chemicals. The dietary uptake of chemicals by predator i from prey x in the screening-level model is defined as:

$$D = (p_{ix}\ CAE\ I_x)\ C_x \qquad (6)$$

and the consumption rate (I) is defined as:

$$I = [(kG + R)/FAE]*(W/D_x/W/D_i)*(f_{Lx}/f_{Li}), \text{ where} \qquad (7)$$

FAE = food assimilation efficiency, and
W/D = wet/dry weight ratio of prey x or predator i,

for all prey organisms (x) consumed. The FAE, W/D, f_L, and p_{ix} are defined for each key organism in Table 4-10.

Metabolism

The metabolic parameters that described the mechanisms of chemical loss from an organism are depuration (k2), faecal excretion (kE), growth (kG), and chemical metabolism rate (kM). The equations for deriving these factors for the screening-level model are listed in Table 4-10, and are based on relationships described by Thomann (1989), Connolly (1991), or Gobas (1993). According to Gobas (1993), the kM is low for highly hydrophobic organic chemicals. For this screening-level assessment, the kM was assumed to be zero for PCBs, pesticides, and PCDD/Fs. For readily metabolizable compounds such as PAHs, the kM was assumed to be 0.99, since nearly all of the PAHs that are ingested are readily metabolized by most aquatic organisms, particularly those organisms that occupy relatively high trophic levels in the food web (Lee et al., 1976; Lech and Bend, 1980; Solbakken and Palmork, 1981; Varanasi and Gmur, 1981; McElroy et al., 1989; Varanasi et al., 1985; McElroy and Sisson, 1989; Niimi and Dookhran, 1989; Broman et al., 1990; Varanasi and Stein, 1991; Clements et al., 1994).

Migration

As stated in the IWP, the seasonal migration of key organisms was accounted for in the model via a migration factor. To be conservative, a migration factor was not incorporated in the model for blue crab, since male crabs may not migrate out of the River and, therefore, may be exposed to chemicals in sediments and food at the Site throughout the year. For striped bass, it was assumed that adults spend most of their lives migrating along the east coast throughout the winter, and to spawning grounds in the Hudson River during the reproductive season and for a period beyond. Since it is highly unlikely that striped bass spawn in the Passaic River, and given the low abundance and diversity of food sources available for predatory fish in the River, it was assumed that their residence should be extremely limited. For the screening-level model, it was assumed that striped bass adults forage at the Site for about one month during the year. For the remainder of the year, it was assumed that striped bass are not exposed to organic chemicals in sediments. Thus, for this analysis, it was assumed that the concentrations of CPC that are present in striped bass are accumulated only from the Site. This is an overly conservative assumption, since contamination of sediments is widespread along the east coast of the U.S., and in the NY/NJ Harbor Estuary (O'Connor and Huggett, 1988; Kennish et al., 1992; NY/NJ HEP, 1993; NOAA, 1994). However, it is not possible to estimate background exposures to chemicals for migratory fish in a screening-level model. Therefore, the potential risks estimated in this screening-level ERA may represent only a small fraction of the potential risks to migratory fish that occur from exposures to contaminants found in areas other than the Site.

4.4.2.3 Model Validation

Model validation was performed by conducting a limited sensitivity analyses for 2,3,7,8-TCDD, for which historical sediment and biological data (from the mid-1980s) were available from the Site. The 95% UCL of the arithmetic mean of the historical Site sediment data were entered into the model to evaluate the validity of the parameters and relationships used to construct the model, particularly the relative feeding preferences, the CAE, the migrations factor for striped bass, and the allometric and empirical relationships used to define a number of parameters.

The results of the model sensitivity analysis, along with the historically measured concentrations of 2,3,7,8-TCDD in key organisms are presented in Table 4-11. The model estimates were within a factor of two of the mean measured concentrations for blue crab and striped bass, and within a factor of four for mummichog. This suggests that the relationships and assumptions used in the model are appropriate for estimating bioaccumulation of chemicals in the Site food web, at least for highly hydrophobic organic compounds.

4.4.2.4 Screening Analysis for Inorganic Chemicals

It appears that only two studies conducted within the NY/NJ Harbor Estuary have concurrently evaluated the concentrations of inorganic chemicals in sediments and fish or crabs. O'Connor and Rachlin (1982) estimated apparent partition coefficients (K_s) for sediments to aquatic organisms, based on datasets for a limited number of metals including Cd, Pb, Cu, Hg, and Zn. The majority of K_s values were similar for most metals with a range of 0.07 to 1.11 for Cd, Cu, Pb and Hg, and a mean of about 0.5. For Zn, the range was substantially higher (0.22 to 24.18), however, with the exception of one apparent outlier, the majority of the Zn values ranges between 0.22 and 1.53 with a mean of about 0.6. Thus, for the most part, the average K_s for metals measured in fish and crabs was about 0.5, which suggests that about 50 percent of the metals concentrations in sediments (for those metals measured in the study) can be accumulated in fish and crabs. No attempt was made to estimate the accumulation of metals in plankton or polychaetes/oligochaetes, since acute sediment toxicity of metals, not bioaccumulation and chronic toxicity, is the primary risk factor to these organisms.

In a more comprehensive study, Hall and Pulliam (1995) evaluated data on Cr, Pb, and Cu in sediments, blue crab, and mummichog, from the Hackensack River. At two sites with substantially different physical sediment characteristics, the mean K_s were similar for both Cr (0.01 and 0.02, respectively) and Pb (0.01 and 0.1, respectively), in both species. The results (K_s) for Cu varied substantially within and between sites and species, ranging between 0.05 and 9.0. However, the majority of the Cu values ranges between 0.05 and 0.5. This is similar to the range reported by O'Connor for both fish and crabs (0.07 to 1.11).

Table 4-11. Sensitivity Analysis of Screening-Level Food Web Model Using Historical Sediment and Biological Data for 2,3,7,8-TCDD from the Site

Organism	Model Output (µg/kg) (a)	Measured Fish/Crab Concentrations (µg/kg) (b)				Historical Sediment Concentration (µg/kg) (c)
		Min.	Mean	Max.	n	1.7 (n=73)
Mummichog	0.34	0.114	0.114	0.114	1	
Blue Crab	0.78	0.48	0.48	0.48	1	
Striped Bass	1.53(d)	1.01(d)	1.81(d)	2.54(d)	6	

(a) Estimated wet weight concentration using historical surface data from IT (1986)
(b) Measured wet weight concentration reported in NJDEP (1985)
(c) 95% UCL of surface sediment data reported in IT (1986)
(d) Lipid normalized concentration - measured values were only reported for fillet samples which were converted to lipid-normalized values to compare to whole body estimates from the food web model

Based on these investigations, a K_s of 0.5 was used to estimate the potential accumulation of metals in mummichog and blue crab from surface sediment concentrations at the Site. These include the inorganic CPC that were not evaluated in either the O'Connor and Rachlin (1982) or Hall and Pulliam (1995) studies, including Al, Sb, As, Ba, Be, Co, Mn, Ni, Se, Ag, Th, Ti, and Va. For these metals, the bioaccumulation may be either under- or overstated from a K_s of 0.5. K_s values of 0.02 and 0.1 were used to estimate the accumulation of Cr and Pb, respectively, based on the apparently low relative bioaccumulation potential of these metals. For migratory striped bass, a K_s value of 0.25 was used to estimate potential accumulation of inorganic chemicals other than Cr and Pb (i.e., the assumed accumulation of inorganic chemicals in striped bass was about 50 percent of that in resident organisms). Because striped bass spend much less than 50 percent of the year at the Site, this may be an overly conservative assumption. However, unlike the food web exposure analysis, there was no way to evaluate the accuracy of an estimated residence time since historical data are not available regarding concentrations of inorganic chemicals in key organisms at the Site.

4.4.2.5 Results of Food Web Model and Inorganic Analysis

The screening-level model runs for each CPC are presented in Appendix G. The summary of the modeling results (i.e., estimated whole body concentrations) are presented in Tables 4-12 through 4-16.

In general, Aroclor PCBs and pesticides appear to accumulate to the greatest extent in aquatic organisms at the Site. The estimated wet weight concentrations of Aroclors 1248 and 1254 in polychaetes/oligochaetes were 138 and 34 µg/kg, respectively. Estimated Aroclor concentrations in fish and crab range from 8.0 to 360 µg/kg. Those for plankton were generally less than 2 µg/kg.

Estimated concentrations of pesticides in polychaetes ranged from 1.03 to 25.4 µg/kg. Those for fish and crab ranged from 0.11 to 65.6 µg/kg. Estimated concentrations in plankton were less than 0.4 µg/kg.

Estimated concentrations of coplanar PCBs in polychaetes ranged from 0.0041 to 9.7 µg/kg. Those for fish and crab ranged from 0.00034 to 16 µg/kg. By contrast, estimated concentrations of PCDD/Fs in polychaetes ranged from 0.00093 to 0.93 µg/kg. Those for fish and crab ranged

Table 4-12. Estimated Concentrations of Dioxins/Furans in Aquatic Organisms from the Passaic River Study Area

Wet Weight Concentrations (µg/kg)

Organism	2,3,7,8-TCDD	1,2,3,7,8-PeCDD	1,2,3,4,7,8-HxCDD	1,2,3,6,7,8-HxCDD	1,2,3,7,8,9-HxCDD	1,2,3,4,6,7,8-HpCDD	OCDD
Phytoplankton	$4.2x10^{-5}$	$1.1x10^{-6}$	$1.4x10^{-6}$	$4.4x10^{-6}$	$2.1x10^{-6}$	$6.8x10^{-5}$	0.0011
Zooplankton	0.0011	$2.8x10^{-5}$	$3.5x10^{-5}$	0.00011	$5.3x10^{-5}$	0.0017	0.028
Mummichog	0.020	0.00038	0.00037	0.0012	0.00056	0.013	0.14
Blue Crab	0.050	0.00092	0.00093	0.0029	0.0014	0.033	0.40
Striped Bass	0.0046	$7.8x10^{-5}$	$7.2x10^{-5}$	0.00023	0.00011	0.0022	0.021
Polychaete/Oligochaete	0.036	0.00093	0.0012	0.0037	0.0018	0.058	0.93

Lipid Normalized Concentrations (µg/kg-lipid)

Organism	Lipid Content	2,3,7,8-TCDD	1,2,3,7,8-PeCDD	1,2,3,4,7,8-HxCDD	1,2,3,6,7,8-HxCDD	1,2,3,7,8,9-HxCDD	1,2,3,4,6,7,8-HpCDD	OCDD
Phytoplankton	0.010	0.0042	0.00011	0.00014	0.00044	0.00021	0.0068	0.11
Zooplankton	0.050	0.021	0.00055	0.00070	0.0022	0.0011	0.034	0.55
Mummichog	0.025	0.81	0.015	0.015	0.047	0.022	0.51	5.5
Blue Crab	0.028	1.8	0.033	0.033	0.10	0.050	1.2	14
Striped Bass	0.050	0.093	0.0016	0.0014	0.0046	0.0022	0.044	0.43
Polychaete/Oligochaete	0.010	3.6	0.093	0.12	0.37	0.18	5.8	93

Wet Weight Concentrations (µg/kg)

Organism	2,3,7,8-TCDF	1,2,3,7,8-PeCDF	2,3,4,7,8-PeCDD	1,2,3,4,7,8-HxCDF	1,2,3,6,7,8-HxCDF	1,2,3,7,8,9-HxCDF	2,3,4,6,7,8-HxCDF	1,2,3,4,6,7,8-HpCDF	1,2,3,4,7,8,9-HpCDF	OCDF
Phytoplankton	$5.2x10^{-6}$	$5.0x10^{-6}$	$1.3x10^{-5}$	0.00014	$2.2x10^{-5}$	$2.5x10^{-6}$	$7.7x10^{-6}$	0.00045	$1.1x10^{-5}$	0.00087
Zooplankton	0.00013	0.00013	0.00033	0.0035	0.00055	$6.3x10^{-5}$	0.00019	0.011	0.00028	0.022
Mummichog	0.0041	0.0032	0.0064	0.048	0.0076	0.00086	0.0026	0.10	0.0025	0.089
Blue Crab	0.012	0.0085	0.016	0.12	0.018	0.0021	0.0064	0.26	0.0063	0.28
Striped Bass	0.0011	0.00080	0.0015	0.010	0.0016	0.00018	0.00055	0.019	0.00045	0.013
Polychaete/Oligochaete	0.0044	0.0042	0.011	0.12	0.019	0.0021	0.0065	0.38	0.0093	0.74

Lipid Normalized Concentrations (µg/kg-lipid)

Organism	Lipid Content	2,3,7,8-TCDF	1,2,3,7,8-PeCDF	2,3,4,7,8-PeCDD	1,2,3,4,7,8-HxCDF	1,2,3,6,7,8-HxCDF	1,2,3,7,8,9-HxCDF	2,3,4,6,7,8-HxCDF	1,2,3,4,6,7,8-HpCDF	1,2,3,4,7,8,9-HpCDF	OCDF
Phytoplankton	0.010	0.00052	0.00050	0.0013	0.014	0.0022	0.00025	0.00077	0.045	0.0011	0.087
Zooplankton	0.050	0.0026	0.0025	0.0065	0.070	0.011	0.0013	0.0039	0.23	0.0055	0.44
Mummichog	0.025	0.16	0.13	0.25	1.9	0.30	0.034	0.11	4.0	0.099	3.5
Blue Crab	0.028	0.41	0.30	0.56	4.2	0.66	0.075	0.23	9.2	0.22	9.8
Striped Bass	0.050	0.022	0.016	0.029	0.20	0.031	0.0036	0.011	0.37	0.0091	0.26
Polychaete/Oligochaete	0.010	0.44	0.42	1.1	12	1.9	0.21	0.65	38	0.93	74

Table 4-13. Estimated Concentrations of PCBs in Aquatic Organisms from the Passaic River Study Area

Wet Weight Concentrations (µg/kg)

Organism	3,3',4,4'-TetraCB	2',3,4,4',5-PentaCB	2,3',4,4',5-PentaCB	2,3,3',4,4'-PentaCB	2,3,4,4',5-PentaCB	3,3',4,4',5-PentaCB	2,3',4,4',5,5'-HexaCB	2,3,3',4,4',5'-HexaCB	2,3,3',4,4',5-HexaCB
Phytoplankton	0.0013	0.00051	0.0057	0.0027	0.00016	3.8×10^{-5}	0.00091	0.00018	0.00058
Zooplankton	0.033	0.013	0.14	0.068	0.0040	0.0010	0.023	0.0045	0.015
Mummichog	0.89	0.61	6.4	3.2	0.19	0.039	0.71	0.15	0.49
Blue Crab	2.4	1.6	16	8.2	0.50	0.099	1.8	0.37	1.2
Striped Bass	0.23	0.15	1.5	0.77	0.047	0.0091	0.15	0.033	0.11
Polychaete/Oligochaete	1.1	0.86	9.7	4.6	0.27	0.064	1.5	0.31	0.98

3,3',4,4',5,5'-HexaCB	2,3,3',4,4',5,5'-HeptaCB	Aroclor 1248	Aroclor 1254
2.4×10^{-6}	0.00023	0.0816	0.0201
6.0×10^{-5}	0.0058	2.04	0.503
0.0017	0.13	125	30.4
0.0041	0.33	360	87.1
0.00034	0.026	33.1	8.01
0.0041	0.39	138	34.1

Lipid Normalized Concentrations (µg/kg-lipid)

Organism	Lipid Content	3,3',4,4'-TetraCB	2',3,4,4',5-PentaCB	2,3',4,4',5-PentaCB	2,3,3',4,4'-PentaCB	2,3,4,4',5-PentaCB	3,3',4,4',5-PentaCB	2,3',4,4',5,5'-HexaCB	2,3,3',4,4',5'-HexaCB	2,3,3',4,4',5-HexaCB
Phytoplankton	0.01	0.13	0.051	0.57	0.27	0.016	0.0038	0.091	0.018	0.058
Zooplankton	0.05	0.65	0.26	2.9	1.4	0.080	0.019	0.46	0.090	0.29
Mummichog	0.025	36	25	260	130	7.7	1.6	28	6.1	20
Blue Crab	0.028	86	57	590	290	18	3.5	63	13	44
Striped Bass	0.05	4.5	3.0	30	15	0.94	0.18	3.1	0.67	2.2
Polychaete/Oligochaete	0.01	110	86	970	460	27	6.4	150	31	98

3,3',4,4',5,5'-HexaCB	2,3,3',4,4',5,5'-HeptaCB	Aroclor 1248	Aroclor 1254
0.00024	0.023	8.16	2.01
0.0012	0.12	40.8	10.1
0.066	5.2	5,010	1,220
0.15	12	12,800	3,110
0.0069	0.51	662	160
0.41	39	13,800	3,410

Table 4-14. Estimated Concentrations of Pesticides in Aquatic Organisms from the Passaic River Study Area

Wet Weight Concentrations (µg/kg)

Organism	Aldrin	alpha-Chlordane	Beta-BHC	Chlordane	4,4'-DDD	4,4'-DDE	4,4'-DDT	Delta-BHC	Dieldrin	Endrin	gamma-Chlordane	Methoxychlor
Phytoplankton	0.0011	0.0021	7.03×10^{-4}	0.00180	0.0150	0.00501	0.0053	6.10×10^{-4}	0.0028	0.00274	0.00242	0.0055
Zooplankton	0.028	0.052	0.0176	0.0450	0.375	0.125	0.13	0.0153	0.070	0.0685	0.0605	0.14
Mummichog	1.7	3.2	0.441	2.75	22.9	6.29	7.1	0.532	0.93	4.20	3.70	7.8
Blue Crab	4.8	9.1	1.43	7.88	65.6	16.7	19	1.64	3.5	12.0	10.6	22
Striped Bass	0.44	0.84	0.110	0.727	6.03	1.56	1.8	0.136	0.22	1.11	0.978	2.0
Polychaete/Oligochaete	1.9	3.5	1.19	3.05	25.4	8.49	9.0	1.03	4.7	4.64	4.10	9.3

Lipid Normalized Concentrations (µg/kg-lipid)

Organism	Lipid Content	Aldrin	alpha-Chlordane	Beta-BHC	Chlordane	4,4'-DDD	4,4'-DDE	4,4'-DDT	Delta-BHC	Dieldrin	Endrin	gamma-Chlordane	Methoxychlor
Phytoplankton	0.01	0.11	0.21	0.0703	0.180	1.50	0.501	0.53	0.0610	0.28	0.274	0.242	0.55
Zooplankton	0.05	0.55	1.0	0.352	0.900	7.50	2.51	2.7	0.305	1.4	1.37	1.21	2.8
Mummichog	0.025	67	127	17.6	110	914	252	285	21.3	37	168	148	310
Blue Crab	0.028	171	325	51.0	281	2,343	596	690	58.4	123	430	378	793
Striped Bass	0.05	8.9	17	2.19	14.5	121	31.2	36	2.72	4.4	22.2	19.6	41
Polychaete/Oligochaete	0.01	186	353	119	305	2,542	849	898	103	475	464	410	932

Table 4-15. Estimated Concentrations of PAHs in Aquatic Organisms from the Passaic River Study Area

Wet Weight Concentrations (µg/kg)

Organism	Acenaphthene	Acenaphthylene	Anthracene	Benzo(a) anthracene	Benzo(a) pyrene	Benzo(b) fluoranthene	Benzo(k) fluoranthene	Chrysene	Dibenzo(a,h) anthracene
Phytoplankton	0.090	0.066	0.11	0.19	0.20	0.20	0.20	0.22	0.076
Zooplankton	2.3	1.7	2.8	4.8	5.0	5.0	5.0	5.5	1.9
Mummichog	1.2	0.66	2.5	8.3	8.1	8.1	8.1	9.6	1.3
Blue Crab	0.95	0.70	1.2	2.1	2.1	2.1	2.1	2.4	0.50
Striped Bass	0.0045	0.0035	0.0072	0.022	0.020	0.020	0.020	0.025	0.0020
Polychaete/Oligochaete	150	110	190	320	340	340	340	370	130

Dibenzofuran	Fluoranthene	Fluorene	Indeno(1,2,3-c,d)pyrene	2-Methyl-naphthalene	Naphthalene	Phenanthrene	Pyrene
0.078	0.42	0.088	0.14	0.085	0.11	0.26	0.39
2.0	11	2.2	3.5	2.1	2.8	6.5	9.8
1.2	16	1.5	3.4	1.0	0.95	6.0	15
0.83	4.6	0.94	1.1	0.90	1.2	2.8	4.3
0.0041	0.043	0.0047	0.0066	0.0043	0.0065	0.017	0.040
130	710	150	240	140	190	440	660

Lipid Normalized Concentrations (µg/kg-lipid)

Organism	Lipid Content	Acenaphthene	Acenaphthylene	Anthracene	Benzo(a) anthracene	Benzo(a) pyrene	Benzo(b) fluoranthene	Benzo(k) fluoranthene	Chrysene	Dibenzo(a,h) anthracene
Phytoplankton	0.01	9.0	6.6	11	19	20	20	20	22	7.6
Zooplankton	0.05	45	33	55	95	100	100	100	110	38
Mummichog	0.025	49	26	102	330	323	323	323	382	51
Blue Crab	0.028	34	25	42	75	76	76	76	86	18
Striped Bass	0.05	0.090	0.070	0.14	0.43	0.41	0.41	0.41	0.50	0.041
Polychaete/Oligochaete	0.01	15,000	11,000	19,000	32,000	34,000	34,000	34,000	37,000	13,000

Dibenzofuran	Fluoranthene	Fluorene	Indeno(1,2,3-c,d)pyrene	2-Methyl-naphthalene	Naphthalene	Phenanthrene	Pyrene
7.8	42	8.8	14	8.5	11	26	39
39	210	44	70	43	55	130	195
49	647	60	136	40	38	240	601
30	164	33	40	32	41	100	152
0.081	0.86	0.094	0.13	0.086	0.13	0.34	0.80
13,000	71,000	15,000	24,000	14,000	19,000	44,000	66,000

Table 4-16. Estimated Concentrations of Semivolatile Compounds in Aquatic Organisms from the Passaic River Study Area

Wet Weight Concentrations (µg/kg)

Organism	Bis(2-ethyl-hexyl) phthalate	Butyl benzyl phthalate	Di-n-butyl phthalate	Di-n-octyl phthalate	1,2,4-Trichloro-benzene
Phytoplankton	1.8	0.067	0.071	0.090	0.075
Zooplankton	45	1.7	1.8	2.3	1.9
Mummichog	69	2.9	3.1	3.9	1.4
Blue Crab	20	0.74	0.78	0.99	0.80
Striped Bass	0.18	0.0076	0.0081	0.010	0.0043
Polychaete/Oligochaete	3,100	110	120	150	130

Lipid Normalized Concentrations (µg/kg-lipid)

Organism	Lipid Content	Bis(2-ethyl-hexyl) phthalate	Butyl benzyl phthalate	Di-n-butyl phthalate	Di-n-octyl phthalate	1,2,4-Trichloro-benzene
Phytoplankton	0.01	180	6.7	7.1	9.0	7.5
Zooplankton	0.05	900	34	36	45	38
Mummichog	0.025	2,700	120	120	160	57
Blue Crab	0.028	703	26	28	35	29
Striped Bass	0.05	3.6	0.15	0.16	0.20	0.086
Polychaete/Oligochaete	0.01	310,000	11,000	12,000	15,000	13,000

from 0.000072 to 0.4 µg/kg. Both coplanar PCB and PCDD/F concentrations in plankton were less than 0.2 µg/kg, with coplanar PCB concentrations generally being over an order of magnitude greater than PCDD/F concentrations.

Estimated concentrations of PAHs in polychaetes ranged from 112 to 712 µg/kg. Those for fish and crab ranged from 0.002 to 16 µg/kg. Estimated concentrations of PAHs in plankton ranged from 0.076 to 11 µg/kg. These data suggest that PAHs are much more bioavailable to planktonic species than pesticides, PCBs, or PCDD/Fs. This is not surprising, given the generally higher water solubility of semivolatiles than that of highly hydrophobic organic compounds. Similar results were found for phthalates and 1,2,4-trichlorobenzene for which estimated concentrations in plankton ranged from 0.067 to 45 µg/kg. Estimated concentrations of these compounds in polychaetes ranged from 114 to 3,051 µg/kg. Those for fish and crabs ranged from 0.0043 to 69 µg/kg.

The results of the inorganic bioaccumulation analysis are presented in Table 4-17. Concentrations in plankton and polychaetes were not estimated, since acute toxicity of metals to these organisms (i.e., the lower trophic levels) is the primary (risk) assessment endpoint of concern, not bioaccumulation. Additionally, since a mechanistic food web model was not used to evaluated trophic transfers of inorganic chemicals, the concentrations in these organisms were not needed to evaluate concentrations in fish and crab.

Estimated wet weight concentrations of inorganic chemicals in fish and crabs ranged from 0.08 to 7300 mg/kg. As previously indicated, these estimates may over- or understate the accumulation of many inorganic chemicals for which sediment to biota partition estimates are not available. However, the high estimated concentrations for many of these chemicals, relative to the estimated concentrations of organic CPC, suggest that exposure to metals in sediments may pose a substantial bioaccumulative risk to fish and blue crab.

The results of the exposure assessment are evaluated and discussed in the risk characterization (Section 4.6), with respect to the ecological effects data that are presented in Section 4.5. The uncertainties associated with the exposure assessment are discussed in Section 4.6.3 (Identification of Uncertainties).

Table 4-17. Results of Inorganic Bioaccumulation Analysis

				Estimated Whole Body Concentration (mg/kg)	
Chemical	**95% UCL of Mean Sediment Concentration**	**Blue Crab/ Mummichog Ks**	**Striped Bass Ks**	**Blue Crab/ Mummichog**	**Striped Bass**
Aluminum	14600	0.5	0.25	7300	3650
Antimony	10	0.5	0.25	5.0	2.5
Arsenic	15	0.5	0.25	7.6	3.8
Barium	229	0.5	0.25	115	57.3
Beryllium	1.2	0.5	0.25	0.62	0.31
Cadmium	7.2	0.5	0.25	3.6	1.8
Chromium	179	0.02	0.01	3.58	1.79
Cobalt	15	0.5	0.25	7.50	3.75
Copper	260	0.5	0.25	130	65
Lead	395	0.1	0.05	39.5	19.8
Manganese	430	0.5	0.25	215	108
Mercury	3.9	0.5	0.25	2.0	1.0
Nickel	65.4	0.5	0.25	32.7	16.4
Selenium	1.6	0.5	0.25	0.80	0.40
Silver	7.1	0.5	0.25	3.6	1.8
Thallium	0.63	0.5	0.25	0.32	0.16
Titanium	493	0.5	0.25	247	123
Vanadium	43.0	0.5	0.25	21.5	10.7
Zinc	628	0.5	0.25	314	157
Dibutyltin	0.335	0.5	0.25	0.168	0.084
Monobutyltin	0.471	0.5	0.25	0.236	0.118

Ks represents the sediment to biota partition estimate for inorganic chemicals

4.5 Ecological Effects Assessment

Consistent with EPA guidance (EPA, 1989, 1991, 1992b, 1994a), the relationships between the CPC in sediment and key organisms, and the potential ecological effects under consideration are evaluated in Section 4.5. As stated in the IWP, the ecological effects under consideration include mortality, impaired growth and development, and reproductive impairment.

The two primary assessment endpoints being evaluated for the Site are mortality of sediment-associated benthic invertebrates and alteration of the benthic community from direct exposure to CPC in sediment, and acute and/or chronic effects in key secondary and tertiary consumers (i.e., mummichog, blue crab, and striped bass) from bioaccumulation of CPC. The objective of the ecological effects assessment is to identify ecotoxicological criteria for comparison to measured sediment concentrations of CPC, and tissue concentrations of bioaccumulative CPC that were estimated from the food web exposure analysis (Section 4.4). To that end, and consistent with EPA guidance (EPA, 1989, 1991, 1992a, 1994a), the ecological effects assessment consists of the following elements: (a) summary of ecotoxicity information from the literature, including results of field and laboratory studies; (b) evaluation of quantitative structure activity relationships (QSARs); and, (c) identification of ecotoxicological criteria for sediments and key organisms.

4.5.1 Evaluation of Ecological Assessment Endpoints

In this Section, the toxicity of CPC in sediments and tissues are evaluated using available data from the scientific literature. The range of effects concentrations compiled from the literature for each chemical or group of chemicals is summarized in ecotoxicological profiles (Appendix H). In addition, the most conservative effects concentrations that are appropriate for the key organisms at the Site are identified for use in the ecological risk characterization (Section 4.6). Consistent with the assessment endpoints for the screening-level ERA, the ecotoxicological profiles primarily focus on concentrations of chemicals or groups of chemicals in tissue[1] and sediment that reportedly have

[1]In the ecological effects assessment and ecotoxicological profiles, tissue concentration refers to the average whole body concentration of a chemical or group of chemicals, unless otherwise specified.

been associated with no observable adverse effects (i.e., NOAEL) or lowest observed adverse effects (i.e., LOAEL) in aquatic organisms. The profiles are also limited to data for key organisms or organisms from phylogenetic groups similar to key organisms at the Site.

The proposed SQG for CPC are presented in Table 4-7. The SQG are based on concentrations of CPC in sediments that have been reported to cause mortality to benthic invertebrates, and to disrupt or alter benthic communities. The lowest reported SQG are assumed to represent the NOAEL for acute toxicity to benthic invertebrates; these are summarized by chemical in Appendix H.

As discussed in the ecotoxicological profiles, there is a paucity of data regarding tissue-based toxicity data for most bioaccumulative organic compounds and inorganic chemicals. In addition, the data that are available are of limited quantity and, in many cases, of questionable quality. To be consistent for chemical compounds within a given group, or within a number of chemical groups that have similar modes of toxic action, QSARs were used to generate conservative estimates of critical tissue concentrations that would be expected to represent the NOAEL or LOAEL for most bioaccumulative chemicals, as described below.

4.5.1.1 Quantitative Structure Activity Relationships for Bioaccumulative Chemicals

Potential toxicity of chemicals to aquatic organisms from bioaccumulation has traditionally been evaluated by comparing a measured concentration of a given chemical in water or sediment to a threshold concentration (in water or sediment) that has been experimentally determined to cause mortality or adverse physiological or morphological effects in aquatic organisms (i.e., LC_{50}, EC_{50}, NOAEL, or LOAEL). When using this approach, no *a priori* assumptions are made regarding the concentration of chemical accumulated in the body of the organism, particularly at the target site of toxic action, that is responsible for the apparent effect(s). Although this approach has several advantages, most notable its ease of implementation, accurate prediction of the accumulation of chemicals from water and sediment is highly site-specific, and is based primarily on the physicochemical properties of the chemical and exposure media.

Given the site specificity of bioaccumulation, it appears to be more logical to evaluate the risks of bioaccumulative chemicals based on the tissue concentration of chemicals that are reported to cause adverse effects. The site-specific factors that regulate bioaccumulation of chemicals can then be accounted for in the exposure assessment, and the magnitude of bioaccumulation can be estimated or measured. The effects assessment can then be based on the known or estimated concentrations of chemicals in key organisms at the site, which can then be compared to critical tissue concentrations of various chemicals that have been shown to cause adverse effects in a variety of species and phylogenetic groups. A number of researchers have suggested that the tissue-residue approach is more appropriate than water- or sediment-effects-based approach for evaluating the potential toxicity of bioaccumulative chemicals (Friant and Henry, 1985; McCarty et al., 1985; Foulkes, 1990; McKim and Schmeider, 1991; Niemi et al., 1991; Calabrese and Baldwin, 1993; EPA, 1993; McCarty et al., 1993; McCarty and MacKay, 1993).

For the screening-level ERA, the effects assessment and risk characterization for bioaccumulative chemicals were conducted using information on whole body concentrations of chemicals or "critical body residues" (CBR) that reportedly illicit adverse effects in aquatic organisms. The CBR method relies on the identification of a whole body concentration of a chemical that has been demonstrated to be associated with an adverse effect at a target organ or system in a variety of aquatic organisms and phylogenetic groups. The use of a CBR is appropriate and necessary, since the identification of a critical effects concentration in a target organ or system is not usually feasible, even if the primary target has been identified (Foulkes, 1990). As a conservative practice, the most sensitive effect endpoint for a chemical or group of chemicals is evaluated to establish a CBR-based NOAEL or LOAEL. In theory, no increased frequency of adverse effects for any endpoint or organ would be expected in exposed organisms if the whole body concentration of the chemical is prevented from reaching the CBR. Comparisons of estimated or measured tissue concentrations in key organisms to CBR ensure that the evaluation considers bioavailability, uptake from food, effects of metabolism, and accumulation kinetics (McCarty and Mackay, 1993).

Based on an evaluation of available toxicity data and QSARs developed from such data, McCarty and Mackay (1993) and McKim and Schnieder (1991) have suggested that toxicity for specific endpoints is associated with a whole body residue of chemical expressed on a molar basis. These authors have also suggested that the "critical" whole body residue (i.e., CBR) for a chemical is similar among various chemicals with the same mode of toxic action, and is similar between

various phylogenetic groups of organisms (e.g., fish and invertebrates). In addition, data compiled by McCarty and Mackay (1993) suggest that the acute to chronic ratio of CBR is consistently about 10:1. For these reasons, the CBR approach is a valuable tool in evaluating and comparing the potential effects of bioaccumulative chemicals in screening-level risk assessments. As a conservative approach, the lowest reported CBR for acute and chronic effects was used as the NOAEL for assessing the potential acute and chronic effects from CPC at the Site.

In using the CBR approach, it is important to consider the chemical's mode of toxic action. Different modes of toxic action are associated with different ranges of CBR. A non-specific mode of toxic action or narcosis has been suggested for semi-volatile organic compounds such as PAHs and phthalate esters with log K_{ow} values of less than about 6.0 (McCarty et al., 1985; McKim and Schnieder, 1991). Body residues associated with narcosis have been estimated to range from 2 to 8 mmol/kg for acute effects and 0.2 to 0.8 mmol/kg for chronic effects. This results in calculated CBR ranging from 23,240 to 2.2×10^5 µg/kg, and 2.3×10^5 to 2.2×10^6 µg/kg, for chronic and acute effects of individual PAHs, respectively. Those for other semivolatiles (phthalate esters and 1,2,4-trichlorobenzene) ranged from 38,840 to 3.1×10^5, and 3.6×10^5 to 3.1×10^6 µg/kg for chronic and acute effects, respectively.

It is important to note that the CBR reported by McCarty and Mackay (1993) for chemicals that are rapidly metabolized or do not leave a readily detectable marker, such as PAHs and phthalate esters, may overstate the concentration of parent compound(s) that causes the expression of the toxic mode of action. For these chemicals, the persistent body residues are substantially lower than the actual amounts of chemical that are absorbed, assimilated, and rapidly metabolized. In many cases, the metabolites of these chemicals may be substantially more toxic than the parent compounds. Thus, CBR for parent compounds may substantially underestimate the risks of these chemicals. Nonetheless, there does not appear to be a way of accounting for this discrepancy in the screening-level ERA.

For 2,3,7,8-TCDD and chemicals with "dioxin-like" effects, such as other 2,3,7,8-substituted PCDD/Fs and coplanar PCBs the CBR range from 3.0×10^{-6} to 4.0×10^{-5} mmol/kg for acute effects, and 1.5×10^{-7} to 1.4×10^{-6} mmol/kg for chronic effects. The chronic CBR are based on growth and survival of aquatic organisms, particularly in early life stages of fish which appear to

be most sensitive to the "dioxin-like" mode of toxicity. This results in calculated CBR ranging from 0.048 to 0.45 µg/kg, and 0.97 to 13 µg/kg, respectively, for chronic and acute effects of 2,3,7,8-TCDD and TCDD toxic equivalents of other PCDD/Fs and coplanar PCBs.

The most sensitive endpoint for pesticide toxicity appears to be effects on the central nervous system (CNS). In particular, it appears that pesticides act as CNS convulsants. The estimated CBR for acute effects of pesticides range from 0.0018 to 0.005 mmol/kg. Based on an acute to chronic ratio of 10:1, the CBR for chronic effects (CNS seizures) range from 0.00018 to 0.0005 mmol/kg (McCarty and Mackay, 1993). This results in calculated CBR ranging from 52 to 205 µg/kg, and 524 to 2,049 µg/kg, for chronic and acute effects of pesticides, respectively.

4.5.2 Identification of Ecotoxicological Criteria for Key Organisms

Available toxicity information for CPC and key organisms were compiled and evaluated, as described in Section 4.5.1. Critical body residues were calculated for bioaccumulative chemicals, including pesticides, PAHs, phthalates esters, and 2,3,7,8-TCDD and "dioxin-like" chemicals (other 2,3,7,8-substituted PCDD/Fs and coplanar PCBs) using QSARs reported by McCarty and Mackay (1993). Because QSARs are not reported for PCB mixtures (i.e., Aroclors) or inorganic chemicals, their CBR were derived, as possible, from the data presented in Appendix H (Ecotoxicological Profiles). For PCBs, unlike most other organic compounds, there are adequate data available to evaluate the most sensitive endpoints and derive tissue-based NOAELs for aquatic organisms. This was not the case for most inorganic chemicals, for which few or no tissue-effects data are reported. Therefore, CBR were generated only for some inorganic chemicals, including aluminum, arsenic, cadmium, chromium, copper, lead, mercury, selenium, zinc, and butyltins. When only an acute or chronic CBR was reported for an inorganic chemical, the other was calculated using an acute to chronic ratio of 10:1. The calculated and reported CBR for the CPC in aquatic organisms are presented in Table 4-18.

4.6 Ecological Risk Characterization

In a comprehensive ecological risk assessment, the risk characterization phase develops quantitative or qualitative estimates of risk by integrating the exposure profile and effects profile. Potential risks are described for each endpoint and the overall ecological impact is determined by

Table 4-18. Calculation of Critical Body Residues for Aquatic Organisms

Chemical	Molecular Weight (g/mol)	Estimated Residue-Acute (mol/kg) (a)		Estimated Residue-Chronic (mol/kg) (a)		Critical Body Residue - Acute (ug/kg)		Critical Body Residue - Chronic (ug/kg)	
		Min	Max	Min	Max	Min	Max	Min	Max
PCDD/Fs									
TCDD, 2,3,7,8-	322	$3.0x10^{-9}$	$4.0x10^{-8}$	$1.5x10^{-10}$	$1.4x10^{-9}$	0.97	13	0.048	0.45
PECDD, 1,2,3,7,8-	356.4								
HxCDD, 1,2,3,4,7,8-	391								
HxCDD, 1,2,3,6,7,8-	391								
HxCDD, 1,2,3,7,8,9-	391								
HpCDD, 1,2,3,4,6,7,8-	425.2								
OCDD	460								
TCDF, 2,3,7,8-	306								
PECDF, 1,2,3,7,8-	340								
PECDF, 2,3,4,7,8-	340								
HxCDF, 1,2,3,4,7,8-	374.87								
HxCDF, 1,2,3,6,7,8-	374.87								
HxCDF, 1,2,3,7,8,9-	374.87								
HxCDF, 2,3,4,6,7,8-	374.87								
HpCDF, 1,2,3,4,6,7,8-	409.31								
HpCDF, 1,2,3,4,7,8,9-	409.31								
OCDF	443.76								
PCBs									
2',3,4,4',5-PentaCB (IUPAC #123)	326.43								
2,3',4,4',5,5'-HexaCB (IUPAC #167)	360.88								
2,3',4,4',5-PentaCB (IUPAC #118)	326.43								
2,3,3',4,4',5'-HexaCB (IUPAC #157)	360.88								
2,3,3',4,4',5,5'-HeptaCB (IUPAC #189)	395.32								
2,3,3',4,4',5-HexaCB (IUPAC #156)	360.88								
2,3,3',4,4'-PentaCB (IUPAC #105)	326.43								
2,3,4,4',5-PentaCB (IUPAC #114)	326.43								
3,3',4,4',5,5'-HexaCB (IUPAC #169)	360.88								
3,3',4,4',5-PentaCB (IUPAC #126)	326.43								
3,3',4,4'-TetraCB (IUPAC #77)	291.99								
Aroclor 1248	299.5					4,500 (b)		100 (b)	
Aroclor 1254	328.4					4,500 (b)		100 (b)	
Pesticides									
Aldrin	365	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	657	1,825	66	183
alpha-Chlordane	409.8	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	738	2,049	74	205
Beta-BHC	290.85	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	524	1,454	52	145
Chlordane (total)	409.8	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	738	2,049	74	205
DDD, 4,4'-	320	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	576	1,600	58	160
DDE, 4,4'-	318	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	572	1,590	57	159
DDT, 4,4'-	354.5	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	638	1,773	64	177
Delta-BHC	290.85	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	524	1,454	52	145
Dieldrin	381	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	686	1,905	69	191
Endrin	381	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	686	1,905	69	191
gamma-Chlordane	409.8	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	738	2,049	74	205
Methoxychlor	345.65	$1.8x10^{-6}$	$5.0x10^{-6}$	$1.8x10^{-7}$	$5.0x10^{-7}$	622	1,728	62	173

Table 4-18. Calculation of Critical Body Residues for Aquatic Organisms

Chemical	Molecular Weight (g/mol)	Estimated Residue-Acute (mol/kg) (a)		Estimated Residue-Chronic (mol/kg) (a)		Critical Body Residue - Acute (ug/kg)		Critical Body Residue - Chronic (ug/kg)	
		Min	Max	Min	Max	Min	Max	Min	Max
PAHs									
Acenaphthene	154.2	0.002	0.008	0.0002	0.0008	310,000	$1.2x10^6$	30,840	120,000
Acenaphthylene	152.2	0.002	0.008	0.0002	0.0008	300,000	$1.2x10^6$	30,440	120,000
Anthracene	178.2	0.002	0.008	0.0002	0.0008	360,000	$1.4x10^6$	35,640	140,000
Benzo(a)anthracene	228.3	0.002	0.008	0.0002	0.0008	460,000	$1.8x10^6$	45,660	180,000
Benzo(a)pyrene	252	0.002	0.008	0.0002	0.0008	500,000	$2.0x10^6$	50,400	200,000
Benzo(b)fluoranthene	252.3	0.002	0.008	0.0002	0.0008	500,000	$2.0x10^6$	50,460	200,000
Benzo(ghi)perylene	276	0.002	0.008	0.0002	0.0008	550,000	$2.2x10^6$	55,200	220,000
Benzo(k)fluoranthene	252.3	0.002	0.008	0.0002	0.0008	500,000	$2.0x10^6$	50,460	200,000
Chrysene	228.3	0.002	0.008	0.0002	0.0008	460,000	$1.8x10^6$	45,660	180,000
Dibenzo(a,h)anthracene	278.4	0.002	0.008	0.0002	0.0008	560,000	$2.2x10^6$	55,680	220,000
Dibenzofuran	168.21	0.002	0.008	0.0002	0.0008	340,000	$1.3x10^6$	33,642	130,000
Fluoranthene	202.3	0.002	0.008	0.0002	0.0008	400,000	$1.6x10^6$	40,460	160,000
Fluorene	116.2	0.002	0.008	0.0002	0.0008	230,000	$9.3x10^5$	23,240	93,000
Indeno(1,2,3-c,d)pyrene	276.3	0.002	0.008	0.0002	0.0008	550,000	$2.2x10^6$	55,260	220,000
2-Methylnaphthalene	142.19	0.002	0.008	0.0002	0.0008	280,000	$1.1x10^6$	28,438	110,000
Phenanthrene	178.2	0.002	0.008	0.0002	0.0008	360,000	$1.4x10^6$	35,640	140,000
Pyrene	202.3	0.002	0.008	0.0002	0.0008	400,000	$1.6x10^6$	40,460	160,000
Semivolatiles									
Bis(2-ethylhexyl)phthalate	391	0.002	0.008	0.0002	0.0008	780,000	$3.1x10^6$	78,200	310,000
Butyl benzyl phthalate	312	0.002	0.008	0.0002	0.0008	620,000	$2.5x10^6$	62,400	250,000
Di-n-butyl phthalate	278.3	0.002	0.008	0.0002	0.0008	560,000	$2.2x10^6$	55,660	220,000
Di-n-octyl phthalate	391	0.002	0.008	0.0002	0.0008	780,000	$3.1x10^6$	78,200	310,000
Trichlorobenzene, 1,2,4-	181.45	0.002	0.008	0.0002	0.0008	360,000	$1.5x10^6$	36,290	140,000
Inorganics (mg/kg) (b)									
Aluminum						8,100		810 (c)	
Antimony						NA		NA	
Arsenic						8.2		3.0	
Barium						NA		NA	
Beryllium						NA		NA	
Cadmium						6.0 (c)		0.6	
Chromium						44.2 (c)		4.42	
Cobalt						NA		NA	
Copper						36 (c)		3.6	
Lead						120 (c)		12.0	
Manganese						NA		NA	
Mercury						10 (c)		1.0	
Nickel						NA		NA	
Selenium						120 (c)		12	
Silver						NA		NA	
Thallium						NA		NA	
Titanium						NA		NA	
Vanadium						NA		NA	
Zinc						3,000 (c)		300	
Dibutyltin						17		6.3	
Monobutyltin						17		6.3	

(a) The concentration in organisms not expected to cause adverse effects
(b) Derived from literature data reported in Appendix H

weight-of-evidence. In perhaps the most critical element of the risk assessment, the ecological significance of the predicted or observed effects is discussed. Finally, the risk characterization phase analyzes the uncertainty associated with each element of the assessment and summarizes overall confidence in the conclusions.

In this screening-level ERA, the ecological risk characterization is limited to a quantitative evaluation of the relative potential risks of CPC to key organisms at the Site. As described earlier, the two primary assessment endpoints being evaluated for the Site are (1) mortality of sediment-associated benthic invertebrates and alteration of the benthic community from direct exposure to CPC in sediment, and (2) acute and/or chronic effects in key secondary and tertiary consumers (i.e., mummichog, blue crab, and striped bass) from bioaccumulation of CPC. To that end, the relative risks of individual chemicals and chemical groups are evaluated and discussed for each endpoint.

4.6.1 Calculation of Ecotoxicological (Hazard) Quotients

To address the bioaccumulation endpoint, the lowest (i.e., most conservative) CBR derived for each CPC was compared to the estimated tissue concentrations for key organisms at the Site. Ecotoxicological quotients were calculated as acute and chronic hazard quotients (HQ) and are presented for each CPC and key species at the secondary and tertiary consumer levels of the food web, including mummichog, blue crab, and striped bass. Consistent with EPA guidance (1989, 1994), the HQ is defined as the concentration of the CPC in the key organism divided by the ecotoxicological effects concentration, in this case the lowest reported acute and chronic CBR. In general, an HQ that is greater than one suggests that potential risks to ecological receptors may exist at the Site (EPA, 1989, 1994a).

The chemical-specific HQ for mummichog, blue crab, and striped bass are presented in Tables 4-19, 20, and 21, respectively. Acute HQ for mummichog, blue crab, and striped bass, are below one for all chemicals except copper (3.6, 3.6, and 1.8, respectively). Similarly, the chronic HQ for mummichog were below one for all CPC except for Aroclor 1248 (1.3), aluminum (9.0), arsenic (2.5), cadmium (6.0), copper (36), mercury (2.0), lead (3.3), and zinc (1.05). The chronic HQ for blue crab exceeded one for Aroclor 1248 (3.6), 4,4'-DDD (1.1), aluminum (9.0), arsenic (2.5), cadmium (6.0), copper (36), mercury (2.0), lead (3.3), and zinc (1.05), and was

Table 4-19. Calculated Hazard Quotients (HQ) for Mummichog at the Site

Chemical	Critical Body Residue - Acute (ug/kg) (a) Min	Critical Body Residue - Chronic (ug/kg) (a) Min	Estimated Concentration (b) Whole Tissue	TCDD TEF	Acute HQ Min	Chronic HQ Min
PCDD/Fs						
TCDD, 2,3,7,8-	0.97	0.048	0.020	1.0	0.021	0.42
PECDD, 1,2,3,7,8-			0.00038	0.5	0.00020	0.0039
HxCDD, 1,2,3,4,7,8-			0.00037	0.1	3.9×10^{-5}	0.00078
HxCDD, 1,2,3,6,7,8-			0.0012	0.1	0.00012	0.0024
HxCDD, 1,2,3,7,8,9-			0.00056	0.1	6.0×10^{-5}	0.0012
HpCDD, 1,2,3,4,6,7,8-			0.013	0.01	0.00013	0.0026
OCDD			0.14	0.001	0.00014	0.0029
TCDF, 2,3,7,8-			0.0041	0.1	0.00042	0.0084
PECDF, 1,2,3,7,8-			0.0032	0.05	0.00017	0.0033
PECDF, 2,3,4,7,8-			0.0064	0.5	0.0033	0.066
HxCDF, 1,2,3,4,7,8-			0.048	0.1	0.0050	0.099
HxCDF, 1,2,3,6,7,8-			0.0076	0.1	0.00078	0.016
HxCDF, 1,2,3,7,8,9-			0.00086	0.1	8.9×10^{-5}	0.0018
HxCDF, 2,3,4,6,7,8-			0.0026	0.1	0.00027	0.0055
HpCDF, 1,2,3,4,6,7,8-			0.10	0.01	0.0010	0.021
HpCDF, 1,2,3,4,7,8,9-			0.0025	0.01	2.6×10^{-5}	0.00051
OCDF			0.089	0.001	9.2×10^{-5}	0.0018
Total PCDD/Fs			NA	NA	0.033	0.66
PCBs						
2',3,4,4',5-PentaCB (IUPAC #123)			0.61	0.001	0.00063	0.013
2,3',4,4',5,5'-HexaCB (IUPAC #167)			0.71	0.001	0.00074	0.015
2,3',4,4',5-PentaCB (IUPAC #118)			6.4	0.001	0.0066	0.13
2,3,3',4,4',5'-HexaCB (IUPAC #157)			0.15	0.001	0.00016	0.0031
2,3,3',4,4',5,5'-HeptaCB (IUPAC #189)			0.13	0.001	0.00013	0.0027
2,3,3',4,4',5-HexaCB (IUPAC #156)			0.49	0.001	0.00051	0.010
2,3,3',4,4'-PentaCB (IUPAC #105)			3.2	0.001	0.0033	0.066
2,3,4,4',5-PentaCB (IUPAC #114)			0.19	0.001	0.00020	0.0039
3,3',4,4',5,5'-HexaCB (IUPAC #169)			0.0017	0.05	8.6×10^{-5}	0.0017
3,3',4,4',5-PentaCB (IUPAC #126)			0.039	0.1	0.0040	0.081
3,3',4,4'-TetraCB (IUPAC #77)			0.89	0.01	0.0092	0.18
Total Coplanar PCBs			NA	NA	0.026	0.51
Aroclor 1248	4,500	100	125	NA	0.028	1.3
Aroclor 1254	4,500	100	30	NA	0.0068	0.30
Total Aroclor PCBs			NA	NA	0.035	1.6
Pesticides						
Aldrin	657	66	1.7	NA	0.0026	0.026
alpha-Chlordane	738	74	3.2	NA	0.0043	0.043
Beta-BHC	524	52	0.44	NA	0.00084	0.0084
DDD, 4,4'-	576	58	23	NA	0.040	0.40
DDE, 4,4'-	572	57	6.3	NA	0.011	0.11
DDT, 4,4'-	638	64	7.1	NA	0.011	0.11
Delta-BHC	524	52	0.53	NA	0.0010	0.010
Dieldrin	686	69	0.93	NA	0.0014	0.014
Endrin	686	69	4.2	NA	0.0061	0.061
gamma-Chlordane	738	74	3.7	NA	0.0050	0.050
Methoxychlor	622	62	7.8	NA	0.013	0.13
Total Pesticides			NA	NA	0.096	0.96

Table 4-19. Calculated Hazard Quotients (HQ) for Mummichog at the Site

Chemical	Critical Body Residue - Acute (ug/kg) (a) Min	Critical Body Residue - Chronic (ug/kg) (a) Min	Estimated Concentration (b) Whole Tissue	TCDD TEF	Acute HQ Min	Chronic HQ Min
PAHs						
Acenaphthene	308,400	30,840	1.2	NA	$3.9x10^{-6}$	$3.9x10^{-5}$
Acenaphthylene	304,400	30,440	0.66	NA	$2.2x10^{-6}$	$2.2x10^{-5}$
Anthracene	356,400	35,640	2.5	NA	$7.1x10^{-6}$	$7.1x10^{-5}$
Benzo(a)anthracene	456,600	45,660	8.3	NA	$1.8x10^{-5}$	0.00018
Benzo(a)pyrene	504,000	50,400	8.1	NA	$1.6x10^{-5}$	0.00016
Benzo(b)fluoranthene	504,600	50,460	8.1	NA	$1.6x10^{-5}$	0.00016
Benzo(ghi)perylene	552,000	55,200	3.1	NA	$5.6x10^{-6}$	$5.6x10^{-5}$
Benzo(k)fluoranthene	504,600	50,460	8.1	NA	$1.6x10^{-5}$	0.00016
Chrysene	456,600	45,660	9.6	NA	$2.1x10^{-5}$	0.00021
Dibenzo(a,h)anthracene	556,800	55,680	1.3	NA	$2.3x10^{-6}$	$2.3x10^{-5}$
Dibenzofuran	336,420	33,642	1.2	NA	$3.7x10^{-6}$	$3.7x10^{-5}$
Fluoranthene	404,600	40,460	16	NA	$4.0x10^{-5}$	0.00040
Fluorene	232,400	23,240	1.5	NA	$6.4x10^{-6}$	$6.4x10^{-5}$
Indeno(1,2,3-c,d)pyrene	552,600	55,260	3.4	NA	$6.1x10^{-6}$	$6.1x10^{-5}$
2-Methylnaphthalene	284,380	28,438	1.0	NA	$3.5x10^{-6}$	$3.5x10^{-5}$
Phenanthrene	356,400	35,640	6.0	NA	$1.7x10^{-5}$	0.00017
Pyrene	404,600	40,460	15	NA	$3.7x10^{-5}$	0.00037
Low Molecular Wt. PAHs	232,400	23,240	14	NA	$6.1x10^{-5}$	0.00061
High Molecular Wt. PAHs	404,600	40,460	81	NA	0.00020	0.0020
Total PAHs	232,400	23,240	95	NA	0.00041	0.0041
Semivolatiles						
Bis(2-ethylhexyl)phthalate	782,000	78,200	69	NA	$8.8x10^{-5}$	0.00088
Butyl benzyl phthalate	624,000	62,400	2.9	NA	$4.7x10^{-6}$	$4.7x10^{-5}$
Di-n-butyl phthalate	556,600	55,660	3.1	NA	$5.5x10^{-6}$	$5.5x10^{-5}$
Di-n-octyl phthalate	782,000	78,200	3.9	NA	$5.0x10^{-6}$	$5.0x10^{-5}$
Total Phthalates			NA	NA	0.00010	0.0010
Trichlorobenzene, 1,2,4-	362,900	36,290	1.4	NA	$3.9x10^{-6}$	$3.9x10^{-5}$
Inorganics (mg/kg)						
Aluminum	8,100	810 (c)	7,300	NA	0.90	9.0
Antimony	NA	NA	5.0	NA	NA	NA
Arsenic	8.2	3.0	7.6	NA	0.93	2.5
Barium	NA	NA	115	NA	NA	NA
Beryllium	NA	NA	0.62	NA	NA	NA
Cadmium	6.0 (c)	0.6	3.6	NA	0.6	6.0
Chromium	44.2 (c)	4.42	3.6	NA	0.081	0.81
Cobalt	NA	NA	7.5	NA	NA	NA
Copper	36 (c)	3.6	130	NA	3.6	36
Lead	120 (c)	12	40	NA	0.33	3.3
Manganese	NA	NA	215	NA	NA	NA
Mercury	10 (c)	1.0	2.0	NA	0.20	2.0
Nickel	NA	NA	33	NA	NA	NA
Selenium	120 (c)	12	0.80	NA	0.0067	0.067
Silver	NA	NA	3.6	NA	NA	NA
Thallium	NA	NA	0.32	NA	NA	NA
Titanium	NA	NA	247	NA	NA	NA
Vanadium	NA	NA	21	NA	NA	NA
Zinc	3,000	300	314	NA	0.10	1.0
Dibutyltin	17	6.3	0.17	NA	0.010	0.027
Monobutyltin	17	6.3	0.24	NA	0.014	0.038
Total Inorganics					6.8	61

(a) The concentration in organisms not expected to cause adverse effects
(b) The estimated concentration from Site food web model
(c) Calculated assuming acute:chronic ratio of 10:1

Table 4-20. Calculated Hazard Quotients (HQ) for Blue Crab at the Site

	Critical Body Residue - Acute (ug/kg) (a)	Critical Body Residue - Chronic (ug/kg) (a)	Estimated Concentration (µg/kg) (b)	TCDD TEF	Acute HQ	Chronic HQ
Chemical	Min	Min	Whole Tissue		Min	Min
PCDD/Fs						
TCDD, 2,3,7,8-	0.97	0.048	0.050	1.00	0.052	1.0
PECDD, 1,2,3,7,8-			0.00092	0.50	0.00048	0.010
HxCDD, 1,2,3,4,7,8-			0.00093	0.10	0.00010	0.0019
HxCDD, 1,2,3,6,7,8-			0.0029	0.10	0.00030	0.0061
HxCDD, 1,2,3,7,8,9-			0.0014	0.10	0.00014	0.0029
HpCDD, 1,2,3,4,6,7,8-			0.033	0.01	0.00034	0.0069
OCDD			0.40	0.001	0.00042	0.0083
TCDF, 2,3,7,8-			0.012	0.10	0.0012	0.024
PECDF, 1,2,3,7,8-			0.0085	0.05	0.00044	0.0088
PECDF, 2,3,4,7,8-			0.016	0.50	0.0082	0.16
HxCDF, 1,2,3,4,7,8-			0.12	0.10	0.012	0.24
HxCDF, 1,2,3,6,7,8-			0.018	0.10	0.0019	0.038
HxCDF, 1,2,3,7,8,9-			0.0021	0.10	0.00022	0.0043
HxCDF, 2,3,4,6,7,8-			0.0064	0.10	0.00067	0.013
HpCDF, 1,2,3,4,6,7,8-			0.26	0.01	0.0027	0.053
HpCDF, 1,2,3,4,7,8,9-			0.0063	0.01	6.0×10^{-5}	0.0013
OCDF			0.28	0.001	0.00029	0.0057
Total PCDD/F			NA	NA	0.082	1.6
PCBs						
2',3,4,4',5-PentaCB (IUPAC #123)			1.6	0.001	0.0017	0.033
2,3',4,4',5,5'-HexaCB (IUPAC #167)			1.8	0.001	0.0018	0.036
2,3',4,4',5-PentaCB (IUPAC #118)			16	0.001	0.017	0.33
2,3,3',4,4',5'-HexaCB (IUPAC #157)			0.37	0.001	0.00038	0.0077
2,3,3',4,4',5,5'-HeptaCB (IUPAC #189)			0.33	0.001	0.00034	0.0067
2,3,3',4,4',5-HexaCB (IUPAC #156)			1.2	0.001	0.0012	0.025
2,3,3',4,4'-PentaCB (IUPAC #105)			8.2	0.001	0.0085	0.17
2,3,4,4',5-PentaCB (IUPAC #114)			0.50	0.001	0.00052	0.010
3,3',4,4',5,5'-HexaCB (IUPAC #169)			0.0041	0.05	0.00021	0.0042
3,3',4,4',5-PentaCB (IUPAC #126)			0.10	0.1	0.010	0.20
3,3',4,4'-TetraCB (IUPAC #77)			2.4	0.01	0.025	0.50
Total Coplanar PCBs			NA	NA	0.066	1.3
Aroclor 1248	4,500	100	360	NA	0.080	3.6
Aroclor 1254	4,500	100	87	NA	0.019	0.87
Total Aroclor PCBs			NA	NA	0.10	4.5
Pesticides						
Aldrin	657	66	4.8	NA	0.0073	0.073
alpha-Chlordane	738	74	9.1	NA	0.012	0.12
Beta-BHC	524	52	1.4	NA	0.0027	0.027
DDD, 4,4'-	576	58	66	NA	0.11	1.1
DDE, 4,4'-	572	57	17	NA	0.029	0.29
DDT, 4,4'-	638	64	19	NA	0.030	0.30
Delta-BHC	524	52	1.6	NA	0.0031	0.031
Dieldrin	686	69	3.5	NA	0.0050	0.050
Endrin	686	69	12	NA	0.018	0.18
gamma-Chlordane	738	74	11	NA	0.014	0.14
Methoxychlor	622	22	22	NA	0.035	1.0
Total Pesticides			NA	NA	0.27	3.4

Table 4-20. Calculated Hazard Quotients (HQ) for Blue Crab at the Site

	Critical Body Residue - Acute (ug/kg) (a)	Critical Body Residue - Chronic (ug/kg) (a)	Estimated Concentration (µg/kg) (b)	TCDD TEF	Acute HQ	Chronic HQ
Chemical	Min	Min	Whole Tissue		Min	Min
PAHs						
Acenaphthene	308,400	30,840	0.95	NA	$3.1x10^{-6}$	$3.1x10^{-5}$
Acenaphthylene	304,400	30,440	0.70	NA	$2.3x10^{-6}$	$2.3x10^{-5}$
Anthracene	356,400	35,640	1.2	NA	$3.3x10^{-6}$	$3.3x10^{-5}$
Benzo(a)anthracene	456,600	45,660	2.1	NA	$4.6x10^{-6}$	$4.6x10^{-5}$
Benzo(a)pyrene	504,000	50,400	2.1	NA	$4.2x10^{-6}$	$4.2x10^{-5}$
Benzo(b)fluoranthene	504,600	50,460	2.1	NA	$4.2x10^{-6}$	$4.2x10^{-5}$
Benzo(ghi)perylene	552,000	55,200	1.0	NA	$1.8x10^{-6}$	$1.8x10^{-5}$
Benzo(k)fluoranthene	504,600	50,460	2.1	NA	$4.2x10^{-6}$	$4.2x10^{-5}$
Chrysene	456,600	45,660	2.4	NA	$5.3x10^{-6}$	$5.3x10^{-5}$
Dibenzo(a,h)anthracene	556,800	55,680	0.50	NA	$9.0x10^{-7}$	$9.0x10^{-6}$
Dibenzofuran	336,420	33,642	0.83	NA	$2.5x10^{-6}$	$2.5x10^{-5}$
Fluoranthene	404,600	40,460	4.6	NA	$1.1x10^{-5}$	0.00011
Fluorene	232,400	23,240	0.94	NA	$4.0x10^{-6}$	$4.0x10^{-5}$
Indeno(1,2,3-c,d)pyrene	552,600	55,260	1.1	NA	$2.0x10^{-6}$	$2.0x10^{-5}$
2-Methylnaphthalene	284,380	28,438	0.90	NA	$3.2x10^{-6}$	$3.2x10^{-5}$
Phenanthrene	356,400	35,640	2.8	NA	$7.8x10^{-6}$	$7.8x10^{-5}$
Pyrene	404,600	40,460	4.3	NA	$1.1x10^{-5}$	0.00011
Low Molecular Wt. PAHs	232,400	23,240	8.3	NA	$3.6x10^{-5}$	0.00036
High Molecular Wt. PAHs	404,600	40,460	22	NA	$5.5x10^{-5}$	0.00055
Total PAHs	232,400	23,240	31	NA	0.00013	0.0013
Semivolatiles						
Bis(2-ethylhexyl)phthalate	782,000	78,200	20	NA	$2.5x10^{-3}$	0.00025
Butyl benzyl phthalate	624,000	62,400	0.74	NA	$1.2x10^{-6}$	$1.2x10^{-5}$
Di-n-butyl phthalate	556,600	55,660	0.78	NA	$1.4x10^{-6}$	$1.4x10^{-5}$
Di-n-octyl phthalate	782,000	78,200	0.99	NA	$1.3x10^{-6}$	$1.3x10^{-5}$
Total Phtalates			NA	NA	$2.9x10^{-5}$	0.00029
Trichlorobenzene, 1,2,4-	362,900	36,290	0.80	NA	$2.2x10^{-6}$	$2.2x10^{-5}$
Inorganics (mg/kg)						
Aluminum	8,100	810 (c)	7,300	NA	0.90	9.0
Antimony	NA	NA	5.0	NA	NA	NA
Arsenic	8.2	3.0	7.6	NA	0.93	2.5
Barium	NA	NA	115	NA	NA	NA
Beryllium	NA	NA	0.62	NA	NA	NA
Cadmium	6.0 (c)	0.6	3.6	NA	0.6	6.0
Chromium	44.2 (c)	4.42	3.6	NA	0.081	0.81
Cobalt	NA	NA	7.5	NA	NA	NA
Copper	36 (c)	3.6	130	NA	3.6	36
Lead	120 (c)	12	40	NA	0.33	3.3
Manganese	NA	NA	215	NA	NA	NA
Mercury	10 (c)	1.0	2.0	NA	0.20	2.0
Nickel	NA	NA	33	NA	NA	NA
Selenium	120 (c)	12	0.80	NA	0.0067	0.067
Silver	NA	NA	3.6	NA	NA	NA
Thallium	NA	NA	0.32	NA	NA	NA
Titanium	NA	NA	247	NA	NA	NA
Vanadium	NA	NA	21	NA	NA	NA
Zinc	3,000 (c)	300	314	NA	0.10	1.0
Dibutyltin	17	6.3	0.17	NA	0.010	0.027
Monobutyltin	17	6.3	0.24	NA	0.014	0.038
Total Inorganics					6.8	61

(a) The concentration in organisms not expected to cause adverse effects
(b) The estimated concentration from Site food web model
(c) Calculated assuming acute:chronic ratio of 10:1

Table 4-21. Calculated Hazard Quotients (HQ) for Striped Bass at the Site

	Critical Body Residue - Acute (ug/kg) (a)	Critical Body Residue - Chronic (ug/kg) (a)	Estimated Concentration (µg/kg) (b)	TCDD TEF	Acute HQ	Chronic HQ
Chemical	Min	Min	Whole Tissue		Min	Min
PCDD/Fs						
TCDD, 2,3,7,8-	0.97	0.048	0.0046	1.0	0.0048	0.095
PECDD, 1,2,3,7,8-			7.8×10^{-5}	0.5	4.0×10^{-5}	0.00081
HxCDD, 1,2,3,4,7,8-			7.2×10^{-5}	0.1	7.5×10^{-6}	0.00015
HxCDD, 1,2,3,6,7,8-			0.00023	0.1	2.4×10^{-5}	0.00048
HxCDD, 1,2,3,7,8,9-			0.00011	0.1	1.1×10^{-5}	0.00023
HpCDD, 1,2,3,4,6,7,8-			0.0022	0.01	2.3×10^{-5}	0.00046
OCDD			0.021	0.001	2.2×10^{-5}	0.00043
TCDF, 2,3,7,8-			0.00108	0.1	0.00011	0.0022
PECDF, 1,2,3,7,8-			0.00080	0.05	4.1×10^{-5}	0.00083
PECDF, 2,3,4,7,8-			0.0015	0.5	0.00078	0.016
HxCDF, 1,2,3,4,7,8-			0.010	0.1	0.0010	0.021
HxCDF, 1,2,3,6,7,8-			0.0016	0.1	0.00017	0.0033
HxCDF, 1,2,3,7,8,9-			0.00018	0.1	1.9×10^{-5}	0.00037
HxCDF, 2,3,4,6,7,8-			0.00055	0.1	5.7×10^{-5}	0.0011
HpCDF, 1,2,3,4,6,7,8-			0.018	0.01	0.00019	0.0037
HpCDF, 1,2,3,4,7,8,9-			0.00045	0.01	4.7×10^{-6}	9.3×10^{-5}
OCDF			0.013	0.001	1.3×10^{-5}	0.00027
Total PCDD/F			NA	NA	0.0073	0.15
PCBs						
2',3,4,4',5-PentaCB (IUPAC #123)			0.15	0.001	0.00016	0.0031
2,3',4,4',5,5'-HexaCB (IUPAC #167)			0.15	0.001	0.00016	0.0032
2,3',4,4',5-PentaCB (IUPAC #118)			1.5	0.001	0.0016	0.031
2,3,3',4,4',5'-HexaCB (IUPAC #157)			0.033	0.001	3.4×10^{-5}	0.00068
2,3,3',4,4',5,5'-HeptaCB (IUPAC #189)			0.026	0.001	2.7×10^{-5}	0.00054
2,3,3',4,4',5-HexaCB (IUPAC #156)			0.109	0.001	0.00011	0.0023
2,3,3',4,4'-PentaCB (IUPAC #105)			0.77	0.001	0.00080	0.016
2,3,4,4',5-PentaCB (IUPAC #114)			0.047	0.001	4.9×10^{-5}	0.00097
3,3',4,4',5,5'-HexaCB (IUPAC #169)			0.00034	0.05	1.8×10^{-5}	0.00035
3,3',4,4',5-PentaCB (IUPAC #126)			0.0091	0.1	0.00094	0.019
3,3',4,4'-TetraCB (IUPAC #77)			0.23	0.01	0.0024	0.048
Total Coplanar PCBs			NA	NA	0.0062	0.12
Aroclor 1248	4,500	100	33	NA	0.0073	0.33
Aroclor 1254	4,500	100	8.0	NA	0.0018	0.080
Total Aroclor PCBs			NA	NA	0.0091	0.41
Pesticides						
Aldrin	657	66	0.44	NA	0.00067	0.0067
alpha-Chlordane	738	74	0.84	NA	0.0011	0.011
Beta-BHC	524	52	0.11	NA	0.00021	0.0021
DDD, 4,4'-	576	58	3.1	NA	0.0053	0.053
DDE, 4,4'-	572	57	1.6	NA	0.0028	0.028
DDT, 4,4'-	638	64	1.8	NA	0.0028	0.028
Delta-BHC	524	52	0.14	NA	0.00027	0.0027
Dieldrin	686	69	0.22	NA	0.00032	0.0032
Endrin	686	69	1.1	NA	0.0016	0.016
gamma-Chlordane	738	74	0.98	NA	0.0013	0.013
Methoxychlor	622	22	2.0	NA	0.0032	0.091
Total Pesticides			NA	NA	0.020	0.26

Table 4-21. Calculated Hazard Quotients (HQ) for Striped Bass at the Site

	Critical Body Residue - Acute (ug/kg) (a)	Critical Body Residue - Chronic (ug/kg) (a)	Estimated Concentration (µg/kg) (b)	TCDD TEF	Acute HQ	Chronic HQ
Chemical	Min	Min	Whole Tissue		Min	Min
PAHs						
Acenaphthene	308,400	30,840	0.0045	NA	$1.5x10^{-8}$	$1.5x10^{-7}$
Acenaphthylene	304,400	30,440	0.0035	NA	$1.1x10^{-8}$	$1.1x10^{-7}$
Anthracene	356,400	35,640	0.0072	NA	$2.0x10^{-8}$	$2.0x10^{-7}$
Benzo(a)anthracene	456,600	45,660	0.022	NA	$4.8x10^{-8}$	$4.8x10^{-7}$
Benzo(a)pyrene	504,000	50,400	0.020	NA	$4.0x10^{-8}$	$4.0x10^{-7}$
Benzo(b)fluoranthene	504,600	50,460	0.020	NA	$4.0x10^{-8}$	$4.0x10^{-7}$
Benzo(ghi)perylene	552,000	55,200	0.0061	NA	$1.1x10^{-8}$	$1.1x10^{-7}$
Benzo(k)fluoranthene	504,600	50,460	0.020	NA	$4.0x10^{-8}$	$4.0x10^{-7}$
Chrysene	456,600	45,660	0.025	NA	$5.5x10^{-8}$	$5.5x10^{-7}$
Dibenzo(a,h)anthracene	556,800	55,680	0.0020	NA	$3.7x10^{-9}$	$3.7x10^{-8}$
Dibenzofuran	336,420	33,642	0.0041	NA	$1.2x10^{-8}$	$1.2x10^{-7}$
Fluoranthene	404,600	40,460	0.043	NA	$1.1x10^{-7}$	$1.1x10^{-6}$
Fluorene	232,400	23,240	0.0047	NA	$2.0x10^{-8}$	$2.0x10^{-7}$
Indeno(1,2,3-c,d)pyrene	552,600	55,260	0.0066	NA	$1.2x10^{-8}$	$1.2x10^{-7}$
2-Methylnaphthalene	284,380	28,438	0.0043	NA	$1.5x10^{-8}$	$1.5x10^{-7}$
Phenanthrene	356,400	35,640	0.017	NA	$4.8x10^{-8}$	$4.8x10^{-7}$
Pyrene	404,600	40,460	0.040	NA	$9.9x10^{-8}$	$9.9x10^{-7}$
Low Molecular Wt. PAHs	232,400	23,240	0.045	NA	$1.9x10^{-7}$	$1.9x10^{-6}$
High Molecular Wt. PAHs	404,600	40,460	0.21	NA	$5.1x10^{-7}$	$5.1x10^{-6}$
Total PAHs	232,400	23,240	0.25	NA	$1.1x10^{-6}$	$1.1x10^{-5}$
Semivolatiles						
Bis(2-ethylhexyl)phthalate	782,000	78,200	0.18	NA	$2.3x10^{-7}$	$2.3x10^{-6}$
Butyl benzyl phthalate	624,000	62,400	0.0076	NA	$1.2x10^{-8}$	$1.2x10^{-7}$
Di-n-butyl phthalate	556,600	55,660	0.0081	NA	$1.5x10^{-8}$	$1.5x10^{-7}$
Di-n-octyl phthalate	782,000	78,200	0.010	NA	$1.3x10^{-8}$	$1.3x10^{-7}$
Total Phtalates			NA	NA	$2.7x10^{-7}$	$2.7x10^{-6}$
Trichlorobenzene, 1,2,4-	362,900	36,290	0.0043	NA	$1.2x10^{-8}$	$1.2x10^{-7}$
Inorganics (mg/kg)						
Aluminum	8,100	810 (c)	3,650	NA	0.45	4.5
Antimony	NA	NA	2.5	NA	NA	NA
Arsenic	8.2	3.0	3.8	NA	0.46	1.3
Barium	NA	NA	57	NA	NA	NA
Beryllium	NA	NA	0.31	NA	NA	NA
Cadmium	6.0 (c)	0.6	1.8	NA	0.3	3.0
Chromium	44.2 (c)	4.42	1.8	NA	0.040	0.40
Cobalt	NA	NA	3.8	NA	NA	NA
Copper	36 (c)	3.6	65	NA	1.8	18
Lead	120 (c)	12	20	NA	0.16	1.6
Manganese	NA	NA	108	NA	NA	NA
Mercury	10 (c)	1.0	1.0	NA	0.098	0.98
Nickel	NA	NA	16	NA	NA	NA
Selenium	120 (c)	12	0.40	NA	0.0033	0.033
Silver	NA	NA	1.8	NA	NA	NA
Thallium	NA	NA	0.16	NA	NA	NA
Titanium	NA	NA	123	NA	NA	NA
Vanadium	NA	NA	11	NA	NA	NA
Zinc	3,000 (c)	300	157	NA	0.052	0.52
Dibutyltin	17	6.3	0.084	NA	0.0049	0.013
Monobutyltin	17	6.3	0.12	NA	0.0069	0.019
Total Inorganics					3.4	30

(a) The concentration in organisms not expected to cause adverse effects
(b) The estimated concentration from Site food web model
(c) Calculated assuming acute:chronic ratio of 10:1

1.0 for 2,3,7,8-TCDD. For striped bass, the chronic HQ exceeded one for aluminum (4.5), arsenic (1.3), cadmium (3.0), copper (18), and lead (1.6). Individually, chemicals other than those with an acute and/or chronic HQ of one or greater are not expected to cause an adverse acute or chronic effect in mummichog, blue crab, or striped bass. As indicated in Tables 4-19, 4-20, and 4-21 the chronic HQs for most chemicals are also substantially below one.

Direct (acute) toxicity to benthic invertebrates exposed to sediments at the Site was also evaluated using HQ. For this endpoint, HQ were calculated as the ratio of the 95% UCL of the mean surface sediment concentration at the Site to the lowest reported benchmark sediment toxicity value. As described earlier, the lowest available SQG were assumed to represent the appropriate sediment toxicity values (Table 4-22). Direct sediment toxicity was evaluated only for those chemicals which have been shown to cause mortality of benthic invertebrates or alterations in the benthic community (i.e., for chemicals which SQG based on direct sediment toxicity) have been developed. These include inorganic chemicals, PAHs and other semivolatiles, some pesticides, and Aroclor mixtures of PCBs. The latter two groups (PCBs and pesticides) have been shown to have direct toxicity to benthic invertebrates, as well as substantial bioaccumulative effects, primarily due to their relatively high concentrations in sediments from contaminated areas. The same is true for inorganic chemicals and semivolatile compounds, although to a much lesser extent. For other bioaccumulative chemicals such as PCDD/Fs and coplanar PCBs, direct toxicity to invertebrates has not been demonstrated; rather, available SQG have been developed based solely on tissue-residue (e.g., bioaccumulative) effects in secondary and tertiary consumers (EPA, 1993; Iannuzzi, 1995).

With the exception of di-n-butyl phthalate, the concentrations of each CPC (for which an SQG was available) exceeded its SQG (i.e., the HQ was greater than one). This suggests that a large number of CPC may cause adverse effects to benthic invertebrates that live in close association with sediments. For this reason, direct toxicity of CPC in sediments appears to pose a substantially greater risk than does bioaccumulation of CPC to key organisms at the Site. These comparisons are further evaluated below.

Table 4-22. Calculated Hazard Quotients (HQ) for Sediment Toxicity of CPC to Benthic Invertebrates

	Proposed Marine Sediment Quality Guidelines									
	NOAA 1995(b)		WSDOE 1991(f)		FDEP, 1993 (j)		Other As	95% UCL	Hazard	% Contribution to
	ER-L(c,d)	ER-M(d,e)	SQC(g,h)	MCL(g,i)	NOEL (k)	PEL (l)	Specified (m)	on the Mean	Quotient	Hazard Index
Inorganics (ppm):										
Aluminum								14,600		
Antimony							2 (a,n)	10	5.0	0.33
Arsenic	8.2	70	57	93	8	64	7.24 (a,q)	15	2.1	0.14
Barium								229		
Beryllium								1.2		
Cadmium	1.2	9.6	5.1	6.7	1	7.5	0.67 (a,q)	7.2	11	0.72
Chromium	81	370	260	270	33 (a)	240	52.3 (q)	179	5.4	0.36
Cobalt								15		
Copper	34	270	390	390	28	170	18.7 (a,q)	260	14	0.93
Lead	46.7	218	450	530	21 (a)	160	30.2 (q)	395	19	1.3
Manganese								430		
Mercury	0.15	0.71	0.41	0.59	0.1 (a)	1.4	0.13 (q)	3.9	39	2.6
Nickel	20.9	51.6					15.9 (a,q)	65.4	4.1	0.28
Selenium							0.2 (a,o)	1.6	8.0	0.54
Silver	1	3.7	6.1	6.1	0.5 (a)	2.5	0.73 (q)	7.1	14	0.95
Thallium								0.63		
Titanium								493		
Vanadium								43.0		
Zinc	150	410	410	960	68 (a)	300	124 (q)	628	9.2	0.62
Organics:										
PCBs (ppb)										
Aroclor 1248							30 (a,r)	816	27	1.8
Aroclor 1254							60 (a,r)	201	3.4	
Total PCBs	22.7	180	120	650	24	260	21.5 (a,q)	1,017	45	3.0
Semivolatiles (ppb)										
Bis(2-ethylhexyl)phthalate			470	780			182 (a,q)	18,000	99	6.6
Butly benzyl phthalate			49 (a)	640				670	14	0.92
Di-n-butyl phthalate			2,200 (a)	17,000				710	0.32	0.022
Di-n-octyl phthalate			580 (a)	45,000				900	1.6	0.10
Trichlorobenzene, 1,2,4-			8.1 (a)	18				750	93	6.2
PAHs (ppb)										
Acenaphthene	16	500	160	570	22	450	6.71 (a,q)	900	134	9.0
Acenaphthylene	44	640	660	660			5.9 (a,q)	660	112	7.5
Anthracene	85.3	1,100	2,200	12,000	85	740	46.9 (a,q)	1,100	23	1.6
Benzo(a)anthracene	261	1,600	1,100	2,700	160	1,300	74.8 (a,q)	1,900	25	1.7
Benzo(a)pyrene	430	1,600	990	2,100	230	1,700	88.8 (a,q)	2,000	23	1.5
Benzo(b)fluoranthene								2,000		
Benzo(k)fluoranthene							490 (a,p)	2,000	4.1	0.27
Benzo(ghi)perylene			310 (a)	780				1,300	4.2	0.28
Chrysene	384	2,800	1,100	4,600	220	1,700	107.8 (a,q)	2,200	20	1.4
Dibenzo(ah)anthracene	63.4	260	120	330	31	320	6.22 (a,q)	760	7.1	0.47
Dibenzofuran			150 (a)	580				780	125	8.4
Fluoranthene	600	5,100	1,600	12,000	380	3,200	113 (a,q)	4,200	28	1.9

Table 4-22. Calculated Hazard Quotients (HQ) for Sediment Toxicity of CPC to Benthic Invertebrates

	Proposed Marine Sediment Quality Guidelines									
	NOAA 1995(b)		WSDOE 1991(f)		FDEP, 1993 (j)		Other As	95% UCL	Hazard	% Contribution to
	ER-L(c,d)	ER-M(d,e)	SQC(g,h)	MCL(g,i)	NOEL (k)	PEL (l)	Specified (m)	on the Mean	Quotient	Hazard Index
Fluorene	19	540	230	790	18 (a)	460	21.2 (q)	880	8	0.5
Indeno(1,2,3-cd)pyrene			340 (a)	880				1,400	78	5.2
Methylnaphthalene, 2-	70	670	380	640			20.2 (a,q)	850	3	0.2
Naphthalene	160	2,100	990	1,700	130	1,100	34.6 (a,q)	1,100	54	3.6
Phenanthrene	240	1,500	1,000	4,800	140	1,200	86.7 (a,q)	2,600	75	5.0
Pyrene	665	2,600	10,000	14,000	290	1,900	153 (a,q)	3,900	45	3.0
Pesticides (ppb)										
Aldrin							5 (a,r)	11	2.2	0.15
beta-BHC							2 (a,r)	7.03	3.5	0.24
Delta-BHC							5 (a,r)	6.10	1.2	0.08
alpha-Chlordane							1 (a,n)	20.8	42	2.8
gamma-Chlordane							1 (a,n)	24.2	48	3.2
DDD, 4,4'-							1.22 (a,q)	150	123	8.2
DDE, 4,4'-	2.2	27			1.7 (a)	130	2 (n)	50.1	29	2.0
DDT, 4,4'-							1 (a,n)	53	53	3.6
Dieldrin							0.72 (a,q)	28	39	2.6
Endosulfan I								5.13		
Endosulfan II								28.3		
Endosulfan sulfate								9.74		
Endrin								27.4		
Endrin aldehyde								11		
Endrin ketone								24.1		
Heptachlor epoxide (exo)								4.99		
Methoxychlor								55		
Hazard Index									1,493	100

a. Minimum reported screening guidelines for a chemical
b. National Oceanic Atmospheric Administration (NOAA) values for marine and estuarine sediments reprorted in Long et al. (1995)
c. Effect range-low
d. Values reported in dry weight
e. Effect range-median
f. Washington State Department of Ecology
g. Organic values normalized to 1 percent organic carbon for Passaic River sediments; inorganic values reported on a dry weight basis
h. Sediment Quality Criteria
i. Minimum cleanup levels developed for Puget Sound
j. Florida Department of Environmental Protection
k. No Observed Effect Level
l. Permissible Effect Level
m. Where more than one other value was available, the minimum reported guideline was selected for consideration
n. ER-L value as reported in Long and Morgan (1990)
o. Amphipod Apparent Effects Threshold (AET) reported in CASWRCB, 1990
p. Benthic Apparent Effects Threshold (AET) reported in CASWRCB, 1990
r. Proposed national sediment quality criteria
s. Low-molecular-weight PAHs
t. High-molecular-weight PAHs
u. The concentration of Total PCBs is defined as the sum of the individual Aroclor mixtures

4.6.2 Development of Hazard Ranking Index

For each assessment endpoint (i.e., direct sediment toxicity and bioaccumulative effects), HQ were derived for the appropriate key organisms at the Site for each individual chemical/compound and for chemical groups. Although the HQ for some individual chemicals may not exceed one, the combined effects from exposure to multiple chemicals is often more significant than any single chemical. For this reason, the acute and chronic HQ from each chemical group were then totalled to derive an acute and chronic hazard index (HI) for the Site. The HI represents the total potential risks to key organisms at the Site for each assessment endpoint, and is evaluated similar to the HQ for individual chemicals/compounds; an HI that is greater than one is indicative of potential risks to ecological receptors from mixtures of CPC at the Site. Finally, the total HQ for each chemical group, key organism, and endpoint were compared and ranked with respect to the HI for the Site (see Section 4.6.3.4 on limitations of HI due to uncertainties associated with additive risks).

The acute HI for bioaccumulative risks of CPC at the Site is 7.0 for mummichog, 7.3 for blue crab, and 3.4 for striped bass (Table 4-23). The chronic HI for bioaccumulative risks of CPC at the Site is 65 for mummichog, 72 for blue crab, and 31 for striped bass (Table 4-24, 4-25, 4-26). By comparison, the HI for direct (acute) toxicity to benthic invertebrates from CPC at the Site is 1493. These values indicate that key organisms exposed to multiple CPC at the Site may experience both acute and chronic effects, and that the most substantial risks at the Site are associated with direct toxicity of sediments to benthic organisms.

To identify the individual chemicals which contribute the greatest potential risks at the site for each assessment endpoint, the HQ for each CPC was compared to the HI to determine the percent contribution to total risk. Similarly, the total HQ for chemical groups were compared to the HI to determine the percent contribution of each chemical group to the total risk for each assessment endpoint. The results suggest that inorganic chemicals are responsible for the greatest percentage of risk for bioaccumulative effects at the Site (Tables 4-23 through 4-27). As a group, inorganics comprise 99, 93, and 97 percent of the acute HI for mummichog, blue crab, and striped bass, respectively, and 97, 85, and 94 percent of the chronic HI, respectively. Only copper has an acute HQ of greater than one for mummichog, blue crab, and striped bass.

Table 4-23. Percent Contribution of CPC to Acute Hazard Index (HI) for Key Organisms

Mummichog	Acute Hazard Quotient (HQ)	% Contribution to Acute HI	Cummulative % Contribution
Copper	3.6	52	52
Arsenic	0.93	13	65
Aluminum	0.90	13	78
Cadmium	0.60	8.6	87
Lead	0.33	4.8	91
Mercury	0.20	2.9	94
Zinc	0.10	1.5	>95
Hazard Index	6.98	>95	

Blue Crab	Acute HQ	% Contribution to Acute HI	Cummulative % Contribution
Copper	3.6	49	49
Arsenic	0.93	13	62
Aluminum	0.90	12	74
Cadmium	0.60	8.2	83
Lead	0.33	4.6	87
Mercury	0.20	2.7	90
DDD, 4,4'-	0.11	1.6	92
Zinc	0.10	1.4	>92
Hazard Index	7.3	>92	

Striped Bass	Acute HQ	% Contribution to Acute HI	Cummulative % Contribution
Copper	1.8	53	53
Arsenic	0.46	14	66
Aluminum	0.45	13	79
Cadmium	0.3	8.7	88
Lead	0.16	4.8	93
Mercury	0.098	2.8	>95
Hazard Index	3.4	>95	

Table 4-24. Percent Contribution of CPC to Chronic Hazard Index (HI) for Mummichog at the Site

Chemical	Chronic Hazard Quotient (HQ)	% Contribution to Chronic HI	Cummulative % Contribution
Copper	36	56	56
Aluminum	9.0	14	70
Cadmium	6.0	9.3	79
Lead	3.3	5.1	84
Arsenic	2.5	3.9	88
Mercury	2.0	3.0	91
PCB-1248	1.3	1.9	93
Zinc	1.05	1.6	94
Chromium	0.81	1.3	>95
Hazard Index	65	>95	

Table 4-25. Percent Contribution of CPC to Chronic Hazard Index (HI) for Blue Crab at the Site

Chemical	Chronic Hazard Quotient (HQ)	% Contribution to Chronic HI	Cummulative % Contribution
Copper	36	50	50
Aluminum	9.0	13	63
Cadmium	6.0	8.4	71
PCB-1248	3.6	5.0	76
Lead	3.3	4.6	81
Arsenic	2.5	3.5	84
Mercury	2.0	2.7	87
Zinc	1.1	1.5	90
DDD, 4,4'-	1.1	1.6	89
TCDD, 2,3,7,8-	1.0	1.5	91
PCB-1254	0.87	1.2	93
Chromium	0.81	1.1	94
3,3',4,4'-TetraCB (IUPAC #77)	0.50	0.69	>94
Hazard Index	72	>94	

Table 4-26. Percent Contribution of CPC to Chronic Hazard Index (HI) for Striped Bass at the Site

Chemical	Chronic Hazard Quotient (HQ)	% Contribution to Chronic HI	Cummulative % Contribution
Copper	18	57	57
Aluminum	4.5	14	72
Cadmium	3.0	10	81
Lead	1.6	5.1	86
Arsenic	1.3	4.0	90
Mercury	0.98	3.1	94
Zinc	0.52	1.7	95
Chromium	0.40	1.3	96
PCB-1248	0.33	1.1	98
TCDD,2,3,7,8-	0.095	0.3	>98
Hazard Index	31	>98	

Table 4-27. Percent Contribution of Chemical Groups to Hazard Index (HI) for Key Organisms

Mummichog	Hazard Quotient Acute	Hazard Quotient Chronic	Percent Contribution to Acute HI	Percent Contribution to Chronic HI
Total Inorganics	6.8	61	97	94
Total Aroclor PCBs	0.035	1.6	0.50	2.4
Total Pesticides	0.096	0.96	1.4	1.5
Total PCDD/Fs	0.033	0.66	0.47	1.02
Total Coplanar PCBs	0.026	0.51	0.37	0.79
Total PAHs	0.00041	0.0041	0.0059	0.0063
Total Phthalates	0.00010	0.0010	0.0015	0.0016
1,2,4-Trichlorobenzene	3.9×10^{-6}	3.9×10^{-5}	5.5×10^{-5}	6.0×10^{-5}
Hazard Index	7.0	65		

Blue Crab	Hazard Quotient Acute	Hazard Quotient Chronic	Percent Contribution to Acute HI	Percent Contribution to Chronic HI
Total Inorganics	6.8	61	93	85
Total Aroclor PCBs	0.10	4.5	1.4	6.2
Total Pesticides	0.27	3.4	3.7	4.7
Total PCDD/Fs	0.082	1.6	1.1	2.3
Total Coplanar PCBs	0.066	1.3	0.91	1.8
Total PAHs	0.00013	0.0013	0.0018	0.0018
Total Phthalates	2.9×10^{-5}	0.00029	0.00040	0.00040
1,2,4-Trichlorobenzene	2.2×10^{-6}	2.2×10^{-5}	3.0×10^{-5}	3.1×10^{-5}
Hazard Index	7.3	72		

Striped Bass	Hazard Quotient (HQ) Acute	Hazard Quotient (HQ) Chronic	Percent Contribution to Acute HI	Percent Contribution to Chronic HI
Total Inorganics	3.4	30	99	97
Total Aroclor PCBs	0.0091	0.41	0.27	1.3
Total Pesticides	0.020	0.26	0.57	0.81
Total PCDD/Fs	0.0073	0.15	0.21	0.47
Total Coplanar PCBs	0.0062	0.12	0.18	0.40
Total PAHs	1.1×10^{-6}	1.1×10^{-5}	3.1×10^{-5}	3.4×10^{-5}
Total Phthalates	2.7×10^{-7}	2.7×10^{-6}	7.9×10^{-6}	8.7×10^{-6}
1,2,4-Trichlorobenzene	1.2×10^{-8}	1.1×10^{-7}	3.5×10^{-7}	3.8×10^{-7}
Hazard Index	3.4	31		

The inorganics that account for greater than one percent of the acute and chronic HI for all three key organisms include copper, arsenic, aluminum, cadmium, chromium, lead, mercury and zinc. Together, however, copper, arsenic and aluminum generally account for about 75 percent or more of the acute and chronic HI for each key organism. There are no organic compounds that account for more than one percent of the acute HI for any of the key organisms. Aroclor 1248 is the only organic compound that accounts for greater than one percent of the chronic HI for mummichog and striped bass at the Site (1.9 and 1.1 percent, respectively). Organic CPC that account for more than one percent of the chronic HI in blue crab include Aroclor 1248 (5.0 percent), 4,4'-DDD (1.6 percent), 2,3,7,8-TCDD (1.5 percent), and Aroclor 1254 (1.2 percent).

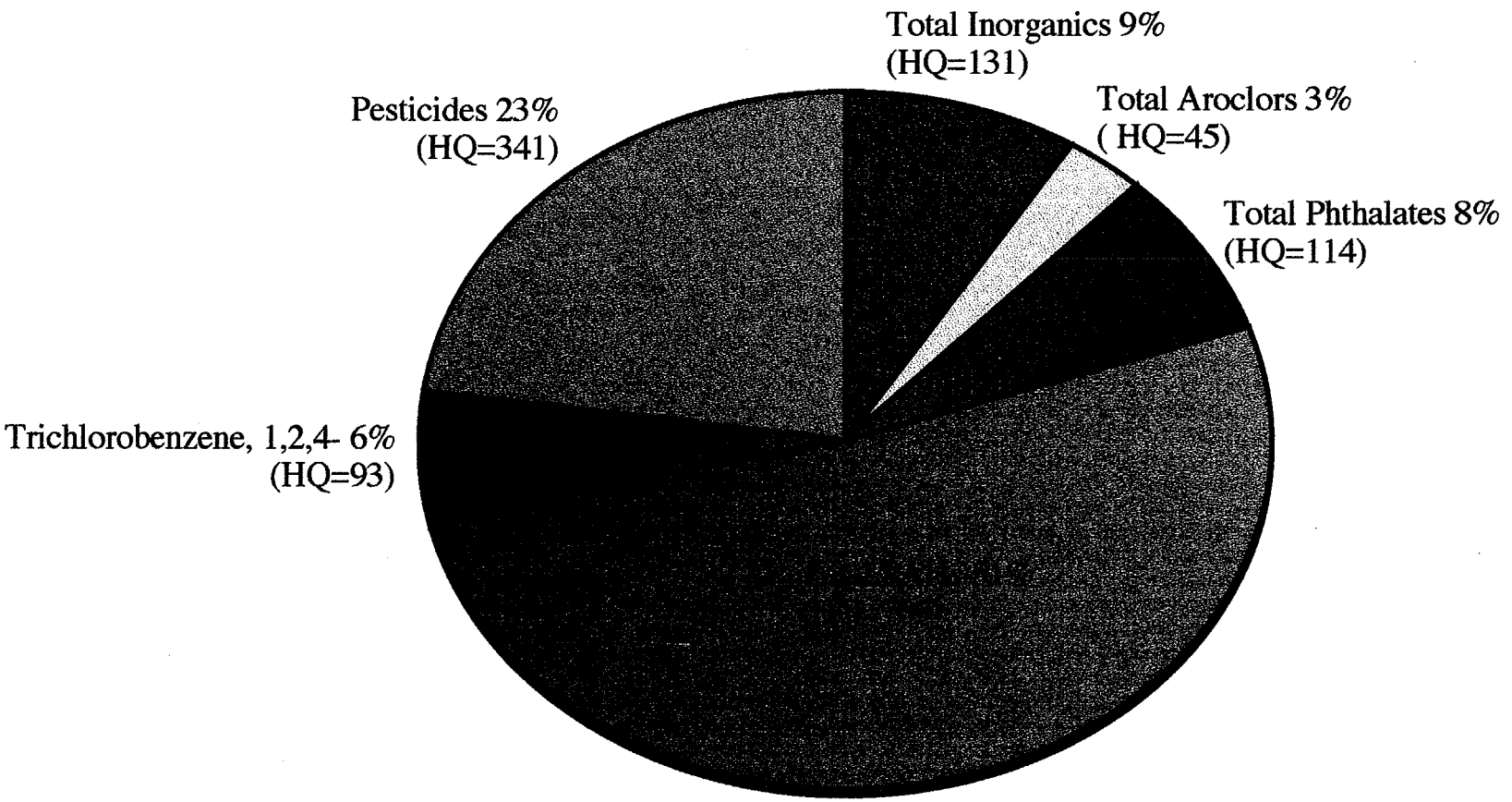
For direct sediment toxicity to benthic organisms, PAHs and pesticides account for 52 and 23 percent of the HI, respectively (Table 4-28; Figure 4-4). Contributions from other chemical groups, including inorganic chemicals, Aroclor PCBs, and other semivolatiles (phthalate esters and 1,2,4-trichlorobenzene) to the HI were generally less than 10 percent for each group. Individually, 23 chemicals (acenaphthene, dibenzofuran, 4,4'-DDD, acenaphthylene, bis(2-ethylhexyl)phthalate, 1,2,4-trichlorobenzene, indeno(1,2,3-cd)pyrene, phenanthrene, naphthalene, 4,4'-DDT, gamma chlordane, pyrene, Aroclors 1248 and 1254 [assessed together as total Aroclor PCBs], mercury, dieldrin, 4,4'-DDE, fluoranthene, benzo(a)anthracene, anthracene, benzo(a)pyrene, chrysene, and lead) each accounted for greater than one percent of the HI, and together accounted for 92 percent of the HI.

The results of the risk characterization clearly demonstrate that the apparent risks from exposure to CPC at the Site are driven by multiple chemicals from a number of chemical groups. Both chronic and acute risks may exist for secondary and tertiary consumers including mummichog, blue crab, and striped bass, from bioaccumulation of multiple CPC, particularly inorganic chemicals. However, the most apparent risks from CPC at the Site appear to be posed by direct exposure of benthic organisms to sediment-bound CPC. For this endpoint, PAHs and pesticides apparently account for the largest portion of the risk.

4.6.3 Identification of Uncertainties

There are several uncertainties associated with the screening-level ERA, many of which can substantially affect the Risk Characterization for the Site. For this reason, it is important to attempt

Figure 4-4. Percent Contribution of Chemical Groups to Hazard Index (HI) for Sediment Toxicity of CPC



Notes:
Total Hazard Index at the Site is 1493.
HQ is total Hazard Quotient for the chemical group.

Table 4-28. Cumulative Percent Contribution to Hazard Index (HI) for Sediment Toxicity of CPC to Benthic Invertebrates

Chemical	95% UCL on the Mean	Hazard Quotient	% Contribution to HI	Cummulative % Contribution
Acenaphthene	900	134	9	9
Dibenzofuran	780	125	8	17
DDD, 4,4'-	150	123	8.2	26
Acenaphthylene	660	112	7.5	33
Bis(2-ethylhexyl)phthalate	18,000	99	6.6	40
Trichlorobenzene, 1,2,4-	750	93	6.2	46
Indeno(1,2,3-cd)pyrene	1,400	78	5.2	51
Phenanthrene	2,600	75	5.0	56
Naphthalene	1,100	54	3.6	60
DDT, 4,4'-	53	53	3.6	63
gamma-Chlordane	24.2	48	3.2	67
Pyrene	3,900	45	3.0	70
Total PCBs	1,017	45	3.0	73
alpha-Chlordane	20.8	42	2.8	75
Mercury	3.9	39	2.6	78
Dieldrin	28	39	2.6	81
DDE, 4,4'-	50.1	29	2.0	83
Fluoranthene	4,200	28	1.9	84
Benzo(a)anthracene	1,900	25	1.7	86
Anthracene	1,100	23	1.6	88
Benzo(a)pyrene	2,000	23	1.5	89
Chrysene	2,200	20	1.4	91
Lead	395	19	1.3	92
Silver	7.1	14	0.95	93
Copper	260	14	0.93	94
Butyl benzyl phthalate	670	14	0.92	95
Cadmium	7.2	11	0.72	95
Zinc	628	9.2	0.62	96
Selenium	1.6	8.0	0.54	97
Fluorene	880	7.8	0.52	97
Dibenzo(ah)anthracene	760	7.1	0.47	98
Chromium	179	5.4	0.36	98
Antimony	10	5.0	0.33	98
Benzo(ghi)perylene	1,300	4.2	0.28	99
Nickel	65.4	4.1	0.28	99
Benzo(k)fluoranthene	2,000	4.1	0.27	99
beta-BHC	7.03	3.5	0.24	99
Methylnaphthalene, 2-	850	2.5	0.17	100
Aldrin	11	2.2	0.15	100
Arsenic	15	2.1	0.14	100
Di-n-octyl phthalate	900	1.6	0.10	100
Delta-BHC	6.10	1.2	0.082	100
Di-n-butyl phthalate	710	0.32	0.022	100
Aluminum	14,600	NA (a)		
Barium	229	NA (a)		
Beryllium	1.2	NA (a)		
Cobalt	15	NA (a)		
Manganese	430	NA (a)		
Thallium	0.63	NA (a)		
Titanium	493	NA (a)		
Vanadium	43	NA (a)		
Benzo(b)fluoranthene	2,000	NA (a)		
Endosulfan I	5.13	NA (a)		
Endosulfan II	28.3	NA (a)		
Endosulfan sulfate	9.74	NA (a)		
Endrin	27.4	NA (a)		
Endrin aldehyde	11	NA (a)		
Endrin ketone	24.1	NA (a)		
Heptachlor epoxide (exo)	4.99	NA (a)		
Methoxychlor	55	NA (a)		
Hazard Index		1,493	>99	

(a) Sediment toxicity values not available for calculating a hazard quotient

to be consistent in making various assumptions during the Analysis (e.g., exposure and effects assessments). This better enables the risk assessor to judge the relative impact of each decision made in the assessment, and evaluate the possible outcomes with respect to the accuracy of the results. From a regulatory perspective, by consistently selecting conservative assumptions throughout the Analysis, the resulting uncertainties in the screening-level ERA should be unidirectional, and the actual risks should be substantially less than those that are calculated. This approach is consistent with EPA guidance (1992a, 1994a), and was used in the screening-level ERA. The uncertainties associated with each phase of the screening-level ERA are summarized and qualitatively discussed below.

4.6.3.1 Selection of Ecological CPC

The selection of CPC for the ecological risk assessment does not take into account the possible contribution of bioaccumulative effects from chemicals with a log K_{ow} of less than 3.5. However, given the relatively low contribution of CPC with log K_{ow} values ranging between 3.5 and 6.0 (e.g., PAHs and other semivolatiles) to the HI for chemicals with potential bioaccumulative effects, it is apparent that this screening criterion is appropriate and conservative. Also conservatism is inherent from including the full list of inorganic CPC as potentially bioaccumulative. However, given the paucity of data regarding the bioaccumulative potential for many of the inorganic CPC at the Site, and the lack of a screening method for inorganic chemicals (similar to that for the organic CPC), it was necessary to assume that each may be bioaccumulated by key organisms. The overall result of the CPC screening was, therefore, a conservative (i.e., large) list of ecological CPC that include many which may pose little or no threat to ecological receptors at the Site. This is further supported by the fact that frequency of detection was not accounted for in the CPC screen and, therefore, many chemicals detected infrequently at the Site are included in the list of ecological CPC.

4.6.3.2 Exposure Assessment

The primary uncertainties associated with the exposure assessment are related to estimating concentrations of chemicals that may be present in key organisms in the absence of available Site-specific data. The use of a food web model to evaluate potential bioaccumulation of organic compounds, and generic sediment-to-biota partition coefficients to evaluate the potential

bioaccumulation of inorganic chemicals is appropriate and has, to some extent, been validated using historical Site sediment and biological data, as well as the results of previous investigations conducted in the NY/NJ Harbor Estuary. However, the estimates of potential bioaccumulation can only be fully validated by collecting Site-specific data regarding the concentrations of CPC in key organisms.

In particular, evaluating the bioaccumulation of inorganic chemicals using empirical relationships such as sediment-to-biota ratios is highly uncertain. However, unlike organic CPC, there are no appropriate models, based on scientifically defensible relationships, that can be used to evaluate the accumulation of inorganic chemicals from sediments and through a food web. Therefore, the use of empirical relationships, based on an evaluation of available data from other Sites, is the only appropriate means to make some estimate of the potential accumulation of inorganic chemicals at various levels of the food web. Although the method used in this analysis is overly simplified, it appear to be, for the most part conservative, in that it likely overstates the potential bioaccumulation of some inorganic chemicals, while understating the accumulation of relatively few chemicals.

The uncertainties associated with the food web model can be divided into two categories: those associated with physicochemical parameters in the model, and assumptions regarding the bioenergetics and ecology of key organisms. The latter include the selection of point estimates for feeding interactions of key organisms (i.e., exposure pathways), the bioenergetics parameters for key organisms (i.e., growth, respiration, excretion, and metabolic rates, and chemical assimilation efficiencies), the reported BSAFs for polychaetes/oligochaetes, and the migration factor for striped bass. For each of these parameters, values or relationships were carefully selected through a review of the ecological community data, and scientific literature regarding the feeding and behavioral ecology of the key organisms. The sensitivity analysis conducted for the model appears to confirm the appropriateness and conservatism of the values that were selected for these parameters, although there are always uncertainties and variability associated with physiological and ecological parameters.

Physicochemical parameters that influence the results of the food web model include the data analysis of concentrations of organic CPC in sediments, the reported partition coefficients for organic CPC (i.e., log K_{ow}), the organic carbon content of the sediments, and the estimated surface water concentrations of organic CPC. The latter is highly uncertain, given the absence of

data regarding concentrations of CPC in surface water at the Site. Nonetheless, the method used to estimate the concentrations of organic CPC in surface water is highly conservative, since it assumes a simple partitioning between sediments and surface water, and does not account for physicochemical factors that substantially limit the concentrations of hydrophobic organic chemicals in water. As a result, the bioconcentration or uptake of chemicals from water by key organisms is likely overstated in the exposure assessment. Likewise, the exposure point concentration of CPC in sediments was expressed as the 95th percent UCL of the arithmetic mean of the Site surface sediment data. This may have resulted in overstated exposures of key organisms to many CPC in sediments and, thus, overstated bioaccumulation. However, the conservatism of the assessment is appropriate for estimating the reasonable maximum exposures in this screening-level ERA, and for evaluating the potential bioaccumulation of CPC in key organisms at the Site. By ensuring that the exposure assessment is conservative, the effects assessment and risk characterization will be inherently conservative as well.

4.6.3.3 Ecological Effects Assessment

The primary uncertainties associated with an ecological effects assessment in a screening-level ERA is the selection of assessment endpoints for consideration, evaluation of the most sensitive effects (i.e., stressor-response) of individual chemicals or chemical groups, and the selection of effects-based concentrations of CPC that will be protective of aquatic organisms. The assessment endpoints considered in the screening-level ERA were direct toxicity of CPC in sediments to benthic organisms that may disrupt or alter benthic communities, and indirect acute and/or chronic effects of CPC that may be bioaccumulated by secondary and tertiary consumers at the Site. Consistent with EPA guidance (1992a, 1994a), these are the primary assessment endpoints that should be evaluated in aquatic ecosystems.

There are a variety of methods for evaluating the potential acute and chronic effects associated with bioaccumulation of chemicals in aquatic systems. Each method is equally uncertain when dealing with mixtures of chemicals. The primary reason is that there is a paucity of data regarding the tissue concentrations of chemicals that are associated with adverse effects in aquatic organisms. Those data that are available are often of questionable quantity and quality. The range of species tested is broadly distributed across a number of phylogenetic groups, as well as habitat types and systems. For these reasons, the extrapolation of such data to organisms or systems that are vastly

different than those being tested are questionable. However, in recent years, there has been increasing consensus among the scientific community that the mechanism of toxicity of many chemicals and chemical groups can be categorized into a limited number of effects endpoints (i.e., narcosis, central nervous system convulsants, early life stage growth/survival impairment, impaired reproductive success, etc.) (McKim and Schmeider, 1991; Calabrese and Baldwin, 1993). In addition, an evaluation of water-based toxicity/bioconcentration data and limited tissue-effects data indicates that the whole body concentrations of chemicals that cause a specific endpoint are similar for various aquatic organisms and phylogenetic groups when expressed on a molar basis. The result has been the development of QSARs, based on various effects endpoints for both acute and chronic effects of chemicals on aquatic organisms. Although there is some uncertainty associated with the use of QSARs, the approach appears to be substantially more sound than relying on the limited tissue-effects data that is available for each individual chemical, or sediment/water effects data for bioaccumulative chemicals. For these reasons, QSARs are the most appropriate method for deriving tissue-based NOAELs for bioaccumulative CPC at the Site.

4.6.3.4 Risk Characterization

To evaluate the sediment toxicity of CPC to benthic organisms, the lowest reported SQG for CPC were directly compared to the 95 percent UCL of the surface sediment data. An HQ was calculated for each CPC, as possible, and a HI was calculated for the assessment endpoint by totalling the HQ for each chemical group. An obvious uncertainty in this approach is the absence of reported SQG for a number of CPC. For this reason, the risk characterization does not take into account many chemicals that may be directly toxic to benthic organisms.

In this screening-level risk assessment, reported QSARs for various chemical groups and effects were used to calculate the lowest whole body tissue-residue (e.g., the CBR) that has been demonstrated to be the threshold for the most sensitive effects of an individual chemical or group of chemicals in aquatic organisms. The QSAR approach was used for pesticides, PCDD/Fs and coplanar PCBs, PAHs, and other semivolatile compounds (phthalate esters and 1,2,4-trichlorobenzene). QSARs have not been reported for Aroclor PCBs or inorganic chemicals. For Aroclor PCBs the most sensitive effects that have been reported in the literature (i.e., impaired reproductive success), and the lowest associated NOAELs for acute and chronic effects were derived from the ecotoxicity data that are compiled and presented in Appendix H (ecotoxicity

profiles). For inorganic chemicals, the limited tissue-effects data that have been reported in the literature were used to derive NOAELs, as possible. Similar to the sediment toxicity assessment, the fact that tissue-effects data are not available for many inorganic chemicals has resulted in some potentially bioaccumulative and toxic chemicals not being evaluated in the risk characterization. For organic compounds, the use of QSARs assures that each CPC is evaluated in the effects assessment and risk characterization.

The lowest calculated CBR was used as the tissue-based NOAEL for the effects assessment. The estimated tissue concentration of each CPC (from either the food web model or empirical analysis for inorganic CPC) in key secondary and tertiary consumers at the Site (i.e., mummichog, blue crab, and striped bass) were directly compared to the NOAEL. An HQ was calculated for each CPC, as possible, and a HI was calculated for the assessment endpoint by totalling the HQ for each chemical group. The use of a NOAEL and estimated tissue concentrations that were based on a reasonable maximum exposure scenario, ensures that the individual HQ are conservative.

The largest uncertainty with using the ecotoxicological quotient (e.g., HQ/HI) approach in a risk assessment is the assumption that the risks from various chemicals in a mixture are additive. Although this is not likely the case, addressing the relative risk potential of chemicals in a mixture is difficult, and beyond the scope of a screening-level ERA.

4.7 Perspective on Ecological Risk

As previously stated, the purpose of this screening-level ecological risk assessment was to evaluate the potential adverse impacts of Site-related CPC and other stressors on key organisms at the Site. As a screening-level ERA, it was appropriate to use conservative approaches in conformance with EPA (1989, 1992a, 1994a) guidance to meet the objectives of the study. However, this screening-level assessment for a complex waterway with multiple sources of contamination is necessarily a limited evaluation. Our analysis, which relies primarily on the use of predictive modeling as well as on information from the published and unpublished literature, was not designed to address the question as to whether there is a significant risk of harm to the overall ecosystem of the Passaic River from the effects of Site-related CPC. Rather, our screening-level assessment focuses on potential effects on individual organisms, and assumes that effects predicted for individuals can result in effects at the population or community level.

While modeling is a useful tool for predicting potential effects, it generally requires substantial extrapolation. Due to the fact that specific data are generally not available for the species of interest, the ecotoxicological data used are usually derived from laboratory studies which must then be extrapolated to natural species. Interspecies extrapolations are known to introduce considerable uncertainty into an analysis. In addition to differences between species, the tremendous variation in natural populations compared with their laboratory counterparts will oftentimes limit the applicability of laboratory results to field situations. Furthermore, conditions in the wild vary considerably from laboratory conditions due to competition, habitat variability, and predation, and, as a result, laboratory data which are obtained under artificial conditions may be of limited use in predicting what will actually occur in the natural ecosystem. In addition, because the input data on toxicity that are required by such models are very limited and are not compiled in a single guidance document or database, it is necessary to conduct a comprehensive literature review, compiling a range of toxicity criteria. These studies and data must then be critically evaluated for applicability to the receptors and stressors of interest at the Site.

It is difficult, if not impossible, to adequately simulate the effects of both competition and predation in the natural environment in predictive risk assessments. The fundamentals of population ecology assert that the health of a community is a function of all interactions within and among species (Begon and Mortimer, 1986), as well as of physical and chemical stressors. The primary types of interactions among species are competition and predation. While predation is often the focus of food web models, competition also plays a critical role in the utilization of limited resources, such as food, water, and breeding territory. By neglecting competition, food web models may substantially over- or underestimate actual exposure. Finally, modeling procedures cannot account for synergistic or antagonistic effects of more than one chemical stressor. Instead, the risk assessor is left with only two options: either consider the effects of two or more stressors to be additive, or ignore the potential effects of the secondary stressor. Either option can lead to over- or underestimates of potential risk.

4.8 Summary and Conclusions

In Section 4.0, a baseline screening-level ERA was performed that evaluates the potential impacts of CPC and other stressors on key organisms at the Site. Consistent with EPA guidance (1992a,

1994a), the potential ecological risks at the Site were evaluated in three stages, consisting of Problem Formulation, Analysis, and Risk Characterization. During Problem Formulation (Section 4.1), the goals and focus of the ecological risk assessment were established. The primary historical stressors that have adversely effected the ecology of the Site are habitat degradation, alteration, and/or removal, and sediment and water quality stressors. The latter include reduced dissolved oxygen and multiple chemical pollutants in sediments and surface waters from a variety of municipal and industrial sources. Consistent with the IWP, the remainder of the screening-level ERA focused on evaluating the potential adverse effects of sediment and water quality stressors on key species at the Site.

Consistent with the second element of EPA's framework (1992a), Analysis, a technical evaluation of data on the potential effects of and exposure to the CPC was performed for key organisms. The analysis consisted of an ecological community characterization (Section 4.2), selection of chemicals of potential concern (Section 4.3), exposure assessment (Section 4.4), and an ecological effects assessment (Section 4.5). Based on an evaluation of the ecological community data for the Site, the key species were determined and a simplified food web was constructed for the Site. The key primary producers at the Site are phytoplankton, while the key primary consumers are zooplankton in the water column, and polychaetes/oligochaetes in sediments. The key secondary consumer at the Site is the mummichog (forage fish). The key tertiary consumers at the Site are striped bass and blue crab, two commercially and recreationally important species on the east coast of the U.S.

The CPC for the Site were selected based on comparisons of sediment concentrations of chemicals to available SQG, and an evaluation of their bioaccumulation potential. The CPC included a number of organic compounds including PCBs, pesticides, PCDD/Fs, PAHs, other semivolatiles, and inorganic chemicals. An exposure assessment was performed to estimate the potential accumulation of organic and inorganic CPC in key organisms at the Site. Consistent with EPA guidance (1992a, 1994a) on conducting screening-level evaluations, conservative assumptions were used in the absence of Site-specific data. To be conservative, the 95 percent UCL of the arithmetic mean of the Site surface sediment data were used as exposure point concentrations in this assessment. The surface sediment data from the biologically active zone (0 to 6 inches) were used to estimate the potential accumulation of chemicals by key organisms from surface sediments and water.

To estimate current concentrations of organic chemicals in key organisms, a screening-level food web exposure analysis was conducted as described in Section 4.4. The analysis considers exposures of key organisms at the Site to chemicals in sediments, surface water, and food sources (i.e., prey). For inorganic chemicals, it is not currently possible to estimate chemical concentrations in aquatic organisms from concentrations in sediments using a mechanistic model. In addition, there are no empirical BAFs published for inorganic chemicals. To that end, estimates of bioaccumulation of inorganic chemicals and organo-metal complexes based on sediment data were made by evaluating the limited data from the scientific literature that presents concurrent measurements of chemical concentrations in sediments and aquatic organisms. From this data estimates of potential partitioning of inorganics between sediments and fish were made.

An ecological effects assessment (Section 4.5) was performed to evaluate and select appropriate sediment- and tissue-based effects concentrations (i.e., NOAELs) for CPC and key organisms at the Site. Finally, in the risk characterization (Section 4.6), the results of the analysis were used to assess the likelihood of adverse impacts associated with the exposure of key organisms to CPC at the Site (EPA 1991, 1992b). The two primary assessment endpoints that were evaluated for the Site are mortality of sediment-associated benthic invertebrates and alteration of the benthic community from direct exposure to CPC in sediment, and acute and/or chronic effects in key secondary and tertiary consumers (i.e., mummichog, blue crab, and striped bass) from bioaccumulation of CPC. To that end, the relative risks of individual chemicals and chemical groups were evaluated and discussed for each endpoint.

The results of the risk characterization suggest that the apparent risks from exposure to CPC at the Site are driven by multiple chemicals from a number of chemical groups. Both chronic and acute risks may exist for secondary and tertiary consumers including mummichog, blue crab, and striped bass, from bioaccumulation of multiple CPC, particularly inorganic chemicals. However, the most apparent risks from CPC at the Site appear to be posed by direct exposure of benthic organisms to sediment-bound CPC. For this endpoint, PAHs and pesticides apparently account for the largest portion of the risk.

4.9 Recommendation

Following the review by Environmental Protection Agency personnel of the detailed basis for, and inherent uncertainties in, the predicted ecological risks presented in this report, a meeting among respondent and agency personnel, in a technical workshop format, would be appropriate to assess the useability of the HERA process and results of this study, including means to reduce the uncertainties in the screening-level assessment. A workshop would serve to focus comments and accelerate reaching a mutual understanding on how to complete a final HERA.

4.10 References for Section 4.0

ACOE. 1980. *Kill Van Kull and Newark Bay Channels, New York and New Jersey, Navigation Study on Improvements to Existing Federal Navigation Channels*. Technical Appendices. U.S. Army Corps. of Engineers, New York District, NY.

ACOE. 1987. *Passaic River Basin, New Jersey and New York Phase I-General Design. Memorandum: Flood Protection Feasibility, Main Stem Passaic River, Main Report and Environmental Impact Statement.* U.S. Army Corps. of Engineers, New York District, NY. December.

ACOE. 1988. *Evaluation of Proposed Dredged Material Disposal Alternatives for New York/New Jersey Harbor, Phase I.* U.S. Army Corps of Engineers. EL-88-11. August.

API. 1972. *Pollution Control on the Passaic River.* American Petroleum Institute, Princeton, NJ.

Begon, M. and M. Mortimer. 1986. *Population Ecology: A Unified Study of Animals and Plants.* Second Edition. Boston: Blackwell Scientific Publications.

Belton, T.J., B.E. Ruppel, and K. Lockwood. 1982. *PCB's in Fish 1975-1980: A Comprehensive Survey.* New Jersey Department of Environmental Protection, Trenton, NJ. November.

Belton, T.J., B. Ruppel, K. Lockwood, and M. Boriek. 1983. *PCBs in Selected Finfish Caught Within New Jersey Waters 1981-1982 (With Limited Chlordane Data).* New Jersey Department of Environmental Protection, Trenton, NJ. July 1.

Belton, T. J., R. Hazen, B.E. Ruppel, K. Lockwood, R. Mueller, E. Stevenson, and J.J. Post. 1985. *A Study of Dioxin (2,3,7,8-Tetrachlorodibenzo-p-Dioxin) Contamination in Selected Finfish, Crustaceans, and Sediments of New Jersey Waterways.* New Jersey Department of Environmental Protection, Office of Science and Research, Trenton, NJ. October 30.

Berg, D.L. and J.S. Levinton. 1985. *The Biology of the Hudson-Raritan Estuary, with Emphasis on Fishes.* National Oceanic and Atmospheric Administration, Rockville, MD. NOAA Technical Memorandum NOS OMA 16. April.

Boesch, D.F. and R.E. Turner. 1984. Dependence of fishery species on salt marshes: The role of food and refuge. *Estuaries* 7:460-468.

Boesch, D.F. and R. Rosenberg. 1981. Response to stress in marine benthic communities. In: *Stress Effects on Natural Ecosystems.* G.W. Barrett and R. Rosenberg (eds.). New York, NY: John Wiley & Sons Ltd. 179-199.

Bonnevie, N.L., D.G. Gunster, and R.J. Wenning. 1992. Lead contamination in surficial sediments from Newark Bay, New Jersey. *Environ. Int.* 18(5):497-508.

Bonnevie, N.L., S.L. Huntley, B.W. Found, and R.J. Wenning. 1994. Trace metal contamination in surficial sediments from Newark Bay, New Jersey. *Sci. Tot. Environ.* 144:1-16.

Brandt, S.B. and K.J. Hartman. 1993. Innovative approaches with bioenergetics models: Future applications to fish ecology and management. *Trans. Am. Fish. Soc.* 122(5):731-735.

Brett, J.R. and T.D.D. Groves. 1979. Chapter 6: Fish physiological energetics. In: *Fish Physiology. Volume III.* Washington, D.C.: Academic Press, Inc. 279-352.

Broman, D., C. Naf, I. Lundbergh, and Y. Zebuhr. 1990. An in situ study on the distribution, biotransformation and flux of polycyclic aromatic hydrocarbons (PAHs) in an aquatic food chain (*seston-mytilus edulis lL. Somateria mollissima L.*) from the Baltic: An ecotoxicological perspective. *Environ. Toxicol. Chem.* 9:429-442.

Brydon, N.F. 1974. *The Passaic River Past, Present, Future.* New Brunswick, NJ: Rutgers University Press.

Burger, J., K. Parsons, and M. Gochfeld. 1993. Avian populations and environmental degradation in an urban river: The kills of New York and New Jersey. In: *Avian Conservation.* J. Jackson (ed.). Madison, WI: University Wisconsin Press.

Bush, B., R.W. Streeter, and R.J. Sloan. 1988. Polychlorobiphenyl (PCB) congeners in striped bass (*Morone saxatilis*) from marine and estuarine waters of New York State determined by capillary gas chromatography. *Arch. Environ. Contam. Toxicol.* 18.

Calabrese, E.J. and L.A. Baldwin. 1993. *Performing Ecological Risk Assessments.* Chelsea, MI: Lewis Publishers.

CASWRCB. 1990. *Evaluation of the AET Approach for Assessing Contamination of Marine Sediments in California.* California Water Resources Control Board, Sacramento, CA. Report No. 90-3WQ. June.

Cerrato, R.M. 1986. *The Benthic Fauna of Newark Bay.* State University of New York, Marine Sciences Research Center, Stony Brook, NY. Special Report 68 and Reference 86-3. April.

Cerrato, R.M. and H.J. Bokuniewicz. 1986. *The Benthic Fauna at Four Potential Containment/Wetlands Stabilization Areas in the New York Harbor Region.* State University of New York, Marine Sciences Research Center, Stony Brook, NY. Special Report 73 and Reference 86-10. August.

ChemRisk. 1995a. *Evaluation of Aquatic and Terrestrial Habitats within the Passaic River Study Area, Appendix E.* Prepared by ChemRisk® A Division of McLaren/Hart, Portland, ME for the Maxus Energy Corporation, Dallas, Texas. January 12.

ChemRisk. 1995b. *Finfish and Benthic Invertebrate Survey of the Passaic River Study Area, Appendix F.* Prepared by ChemRisk® A Division of McLaren/Hart, Portland, ME for the Maxus Energy Corporation, Dallas, Texas. January 18.

Clayton, J.R., S.P. Pavlou, and N.F. Breitner. 1977. Polychlorinated biphenyls in coastal marine zooplankton: Bioaccumulation by equilibrium partitioning. *Environ. Sci. Technol.* 11(7):676-682.

Clements, W.H., J.T. Oris, and T.E. Wissing. 1994. Accumulation and food chain transfer of fluoranthene and benzo(a)pyrene in *Chironomus riparius* and *Lepomis macrochirus*. *Arch. Environ. Contam. Toxicol.* 26:261-266.

Connolly, J.P. 1991. Application of a food chain model to polychlorinated biphenyl contamination of the lobster and winter flounder food chains in New Bedford Harbor. *Environ. Sci. Technol.* 25(4):760-770.

Connolly, J.P. and R. Tonelli. 1985. Modelling kepone in the striped bass food chain of the James River estuary. *Est. Coast. Shelf Sci.* 20:349-357.

Connolly, J.P. and C.J. Pedersen. 1988. A thermodynamic-based evaluation of organic chemical accumulation in aquatic organisms. *Environ. Sci. Technol.* 22(1):99-103.

Courtney, W.A.M. and W.J. Langston. 1978. Uptake of polychlorinated biphenyl (Aroclor 1254) from sediment and from seawater in two intertidal polychaetes. *Environ. Pollut.* 15:303.

Crawford, D.W., N.L. Bonnevie, C.A. Gillis, and R.J. Wenning. 1994. Historical changes in the ecological health of the Newark Bay Estuary, New Jersey. *Ecotoxicol. Environ. Saf.* 29:276-303.

Crawford, D.W., N.L. Bonnevie, and R.J. Wenning. 1995. Sources of pollution and sediment contamination in Newark Bay, New Jersey. *Ecotox. Environ. Saf.* 30:85-100.

Cristini, A. 1991. *Synthesis of Information on the Distribution of Benthic Invertebrates in the Hudson/Raritan System.* Final Report. Ramapo College of New Jersey, Mahwah, N.J. Grant # 12330036-0.

Cunningham, J.T. 1966a. *New Jersey: America's Main Road.* New York, NY: Doubleday & Company.

Cunningham, J.T. 1966b. *Newark.* Newark, NJ: New Jersey Historical Society.

Day, J.W., C.A.S. Hall, W.M. Kemp, and A.Yanez-Arancibia. 1989. *Estuarine Ecology.* New York, NY: John Wiley & Sons.

Earll, E. 1887. Part VII. New Jersey and its fisheries. In: *The Fisheries and Fishery Industries of the United States.* G.B. Goode (ed.). Washington, D.C.: U.S. Government Printing Office.

Ebasco Services Incorporated. 1993. *Remedial Investigation for Tabbs Creek, Langley Research Center, Hampton, Virginia - Draft Report.* NASA Contract No. NASW-4598, NASA Task Assignment No. 9. February.

EPA. 1982. *Aquatic Fate Process Data for Organic Priority Pollutants; Final Report.* Prepared by SRI International, Menlo Park, CA for the U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, D.C. PB87-169090. December.

EPA. 1989. *Risk Assessment Guidance for Superfund; Volume II: Environmental Evaluation Manual - Interim Final.* U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-89-001. March.

EPA. 1991. *Summary Report on Issues in Ecological Risk Assessment.* Assembled by the Eastern Research Group, Inc., Arlington, MA for the U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, D.C. EPA Contract No. 68-C8-0036. February.

EPA. 1992a. *Framework for Ecological Risk Assessment.* U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, D.C. EPA/630/R-92/001. February.

EPA. 1992b. *Final Guidelines for Exposure Assessment; Notice.* U.S. Environmental Protection Agency, Washington, D.C., 57 Federal Register 104: 22888-22938. May 29.

EPA. 1993. *Technical Basis for Deriving Sediment Quality Criteria for Nonionic Organic Contaminants for the Protection of Benthic Organisms by Using Equilibrium Partitioning.* U.S. Environmental Protection Agency, Office of Water, Washington, D.C. EPA-822-R-93-011. September.

EPA. 1994a. *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments.* U.S. Environmental Protection Agency, Environmental Response Team, Edison, NJ. September 26.

EPA. 1994b. *Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors.* U.S. Environmental Protection Agency, Office of Science and Technology, Washington, DC. PB94-202009. July.

EPA. 1994c. *Estimating Exposure to Dioxin-Like Compounds, Volume 2: Properties, Sources, Occurrence and Background Exposures.* U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC. EPA/600/6-88/005Cb. June.

ERM. 1990. *Risk Assessment of Brodhead Creek Site, Stroudsburg, PA.* Environmental Resources Management, Inc., Exton, PA. Report No. 253-45-00-01. May.

ERM. 1992. *Hilton-Davis ECRA Report.* Environmental Resource Management Group. Prepared for the New Jersey Department of Environmental Protection and Energy, Trenton, NJ.

Esser, S.C. 1982. Long-term changes in some finfishes of the Hudson-Raritan estuary. In: *Ecological Stress and the New York Bight: Science and Management.* G.F. Mayer (ed.). Rockville, MD: National Oceanic and Atmospheric Administration.

Festa, P. and S.J. Toth. 1976. Marshes, mudflats and industry. *New Jersey Outdoors* 3(4):117-122.

Finley, B., R.J. Wenning, M.J. Ungs, S. Huntley, and D.J. Paustenbach. 1990. PCDDs and PCDFs in surficial sediments from the lower Passaic River and Newark Bay. Submitted to Dioxin '90 Conf. Proceedings.

Fordham, C.L. and D.P. Reagan. 1991. Pathways analysis method for estimating water and sediment criteria at hazardous waste sites. *Environ. Toxicol. Chem.* 10:949-960.

Foulkes, E.C. 1990. The concept of critical levels of toxic heavy metals in target tissues. *Crit. Rev. Toxicol.* 20(5):327-339.

Fowler, S.W., G.G. Polikarpov, D.L. Elder, P. Parsi, and J.P. Villeneuve. 1978. Polychlorinated biphenyls: Accumulation from contaminated sediments and water by the polychaete *Nereis diversicolor. Mar. Biol.* 48:303-309.

Franz, D.R. 1982. A historical perspective on mollusks in Lower New York Harbor, with emphasis on oysters In: *Ecological Stress and the New York Bight: Science and Management* G. F. Mayer (ed.). Rockville, MD: National Oceanic and Atmospheric Administration.

Friant, S.L. and L. Henry. 1985. Relationship between toxicity of certain organic compounds and their concentrations in tissues of aquatic organisms: A perspective. *Chemosphere* 14(11/12):1897-1907.

Galishoff, S. 1988. *Newark: The Nation's Unhealthiest City, 1832-1895.* New Brunswick, NJ: Rutgers University Press.

Gillis, C.A., N.L. Bonnevie, and R.J. Wenning. 1993. Mercury contamination in the Newark Bay estuary. *Ecotoxicol. Environ. Saf.* 25:214-226.

Gillis, C.A., N.L. Bonnevie, S.H. Su, J.G. Ducey, S.L. Huntley, and R.J. Wenning. 1995. DDT, DDD, and DDE contamination of sediment in the Newark Bay Estuary, New Jersey. *Arch. Environ. Contam. Toxicol.* 28:85-92.

Gobas, F.A.P.C. 1993. A model for predicting the bioaccumulation of hydrophobic organic chemicals in aquatic food-webs: Application to Lake Ontario. *Ecol. Model.* 69:1-17.

Gobas, F.A.P.C., J.M. Lahittete, G. Garofalo, W.Y. Shiu, and D. Mackay. 1988. A novel method for measuring membrane-water partition coefficients of hydrophobic organic chemicals: Comparison with 1-octanol-water partitioning. *J. Pharmaceut. Sci.* 77(3):265-272.

Greig, S.A., J.C. Coulson and P. Monghan. 1983. Age-related differences in foraging success in the herring gull (*Larus argentatus*). *Animal Behavior* 31(4):1237-1243.

Hall, W.S. and G.W. Pulliam. 1995. An assessment of metals in an estuarine wetlands ecosystem. *Arch. Environ. Contam. Toxicol.* (submitted)

Hauge, P., J. Bukowski, P. Morton, M. Boriek, and G. Casey. 1990. Polychlorinated Biphenyls (PCBs), *Chlordane, and DDTs in Selected Fish and Shellfish from New Jersey Waters, 1986-1987: Results from New Jersey's Toxics in Biota Monitoring Program.* New Jersey Department of Environmental Protection, Trenton, NJ.

Hauge, P., J. Bukowski, P. Morton, M. Boriek, and G. Casey. 1993. *Polychlorinated Biphenyls (PCBs), Chlordane, and DDTs in Selected Fish and Shellfish from New Jersey Waters, 1988-1991: Results from New Jersey's Toxics in Biota Monitoring Program.* New Jersey Department of Environmental Protection, Trenton, NJ.

HSDB. 1995. *Chemical Search.* Hazardous Systems Data Base, Betheseda, MD.

Huntley, S.L., N.L. Bonnevie, R.J. Wenning, and H. Bedbury. 1993. Distribution of polycyclic aromatic hydrocarbons (PAHs) in three Northern New Jersey Waterways. *Bull. Environ. Contam. Toxicol.* 51:865-872.

Huntley, S.L., N.L. Bonnevie, and R.J. Wenning. 1995. Polycyclic aromatic hydrocarbon and petroleum hydrocarbon contamination in sediment from the Newark Bay Estuary, New Jersey. *Arch. Environ. Contam. Toxicol.* 28:93-107.

Hurley, A. 1992. Oil and Water. *Seaport.* 26(2):14-21.

HydroQual. 1991. *Task 7.1 Assessment of Pollutant Loadings to New York - New Jersey Harbor: Appendices.* HydroQual, Inc., Mahwah, NJ. Job Number: WOCLO302. January.

Iannuzzi, T.J., S.L. Huntley, N.L. Bonnevie, B.L. Finley, and R.J. Wenning. 1995. Distribution and possible sources of polychlorinated biphenyls in dated sediments from the Newark Bay Estuary, New Jersey. *Arch. Environ. Contam. Toxicol.* 28:108-117.

Iannuzzi, T.J. 1991. Unpublished data.

ISC. 1939. *State of New York and State of New Jersey Annual Report of the Interstate Sanitation Commission for the Year of 1939.* Interstate Sanitation Commission, New York, NY.

IT. 1986. *Remediation Report: Parkway Medians, Newark, New Jersey.* International Technology Corporation, Torrance, CA. March.

Kay, D.G. and A.E. Brafield. 1972. *The energy relations of the polychaete neanthes (Nereis) Virens (SARS).* Coastal Ecology Research Station, Nature Conservancy, Colney Lane, Norwich.

Keeton, W.T. 1980. *Biological Science, Third Edition.* New York, NY: W.W. Norton & Company.

Keller, A.A. and K.S. Squibb. 1992. *New York-New Jersey Harbor Estuary Program. Module 4: Nutrient and Organic Loading Review of Effects and Interactions of Hypoxia and Toxic Chemicals.* Final Report. U.S. Environmental Protection Agency, Region II, Boston, MA. June.

Kennish, M.J., T.J. Belton, P. Hauge, K. Lockwood, and B.E. Ruppel. 1992. Polychlorinated biphenyls in estuarine and coastal marine waters of New Jersey: A review of contamination problems. *Rev. Aquat. Sci.* 6(3/4):275-293.

Kneib, R.T. and A.E. Stiven. 1978. Growth, reproduction, and feeding of *Fundulus heteroclitus* (L.) on a North Carolina salt marsh. *J. Exp. Mar. Biol. Ecol.* 31:121-140.

Kneib, R.T. and A.E. Stiven. 1982. Benthic invertebrate responses to size and density manipulations of the common mummichog, *Fundulus heteroclitus*, in an intertidal salt marsh. *Ecology* 63(5):1518-1532.

Laughlin, R.A. 1982. Feeding habits of the blue crab, *Callinectes sapidus rathbun*, in the Apalachicola Estuary, Florida. *Bull. Mar. Sci.* 32(4):807-822.

Lech, J.J. and J.R. Bend. 1980. Relationship between biotransformation and the toxicity and fate of xenobiotic chemicals in fish. *Environ. Health. Perspect.* 34:115-131.

Lee, R.F., C. Ryan, and M.L. Neuhauser. 1976. Fate of petroleum hydrocarbons taken up from food and water by the blue crab, *Callinectes sapidus*. *Mar. Biol.* 37:363-370.

Levinton, J.S. 1982. *Marine Ecology.* Englewood Cliffs, NJ: Prentice-Hall, Inc.

Long, E.R. and L.G. Morgan. 1991. *The Potential for Biological Effects of Sediment-Sorbed Contaminants Tested in the National Status and Trends Program.* NOS OMA 52. National Oceanic and Atmospheric Administration, Seattle, WA.

Long, E.R., D.D. MacDonald, S.L. Smith, and F.D. Calder. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. *Environ. Manage.* 19(1):81-97.

MacDonald, D.D. 1993. *Development of an Approach to the Assessment of Sediment Quality in Florida Coastal Waters*. MacDonald Environmental Sciences Ltd., Ladysmith, British Columbia. January.

MacLean, A.A.E. 1986. Age-specific foraging ability and the evolution of deferred breeding in three species of gulls. *Wilson Bull.* 98(2):267-279.

Manooch, C.S. 1973. Food habits of yearling and adult striped bass, *Morone saxatilis* (Walbaum), from Albemarle Sound, North Carolina. *Chesapeake Science* 14(2):73-86.

McCarthy, R.M. 1969. The carbohydrate metabolism of the clam, *Mya arenaria*, under aerobic and anaerobic conditions. Ph.D. Dissertation, Georgetown University, Washington, D.C. 1-93.

McCarty, L.S. and D. Mackay. 1993. Enhancing and ecotoxicological modeling and assessment. *Environ. Sci. Technol.* 27 (9):1719-1728.

McCarty, L.S., P.V. Hodson, G.R. Craig, and K.L.E. Kaiser. 1985. The use of quantitative structure-activity relationships to predict the acute and chronic toxicity of organic chemicals to fish. *Environ. Toxicol. Chem.* 4:595-606.

McCarty, L.S., D. Mackay, A.D. Smith, G.W. Ozburn, and D.G. Dixon. 1993. Residue-based interpretation of toxicity and bioconcentration QSARs from aquatic bioassays: Polar narcotic organics. *Ecotoxicol. Environ. Saf.* 25:253-270.

McCormick, J.M. and P.T. Quinn. 1975. *Life in Newark Bay.* 12-14.

McCormick, J.M, R.I. Hires, G.W. Luther, and S.L. Cheng. 1983. Partial recovery of Newark Bay, NJ, following pollution abatement. *Mar. Poll. Bull.* 14(5):188-197.

McElroy, A.E. and J.D. Sisson. 1989. Trophic transfer of benzo[a]pyrene metabolites between benthic marine organisms. *Mar. Enivron. Res.* 28:265-269.

McElroy, A.E., J.W. Farrington, and J.M. Teal. 1989. Bioavailability of polycyclic aromatic hydrocarbons in the aquatic environment. In: *Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment.* U. Varanasi (ed.). Boca Raton, FL: CRC Press. 1-39.

McKim, J.M. and P.K. Schmieder. 1991. Bioaccumulation: Does it reflect toxicity. In: *Bioaccumulation in Aquatic Ecosystems: Contributions to Assessment, Proceeding of an International Workship, Berlin.* Weinheim, NY: Verlagsgesell schaft. 161-188.

McLeese, D.W., C.D. Metcalfe, and D.S. Pezzack. 1980. Uptake of PCBs from sediment by *Nereis virens* and *Crangon septemspinosa. Arch. Environ. Contam. Toxicol.* 9:507-518.

Meyers, W.S. 1945. *The Story of New Jersey. Vols.1-4.* New York, NY: Lewis Historical Publishing.

Meyerson, A.L., G.W. Luther, J. Krajewski, and R. Hires. 1981. Heavy metal distribution in Newark Bay sediments. *Mar. Poll. Bull.* 12(7):244-250.

Mytelka, A.I., J.S. Czachor, W.B. Guggino, and H. Golub. 1973. Heavy metals in wastewater and treatment plant effluents. *WPCF* 45(9):1859-1864.

Mytelka, A.I., M. Wendell, P.L. Sattler, and H. Golub. 1981. *Water Quality of the Hudson-Raritan Estuary.* National Oceanic and Atmospheric Administration, Boulder, CO. NOAA Grant #NA80RAD00034.

Mueller, J.A., T.A. Gerrish, and M.C. Casey. 1982. *Contaminant Inputs to the Hudson-Raritan Estuary.* National Oceanic and Atmospheric Administration, Rockville, MD. Report No. OMPA-21. August.

Niemi, G.J., S.P. Bradbury, and J.M. McKim. 1991. The use of fish physiology literature for predicting fish acute toxicity syndromes. In: *Aquatic Toxicology and Risk Assessment: Fourteenth Volume, ASTM STP 1124.* M.A. Mayes and M.G. Barron (eds.). Philadelphia, PA: American Society for Testing and Materials. 245-260.

Niimi, A.J. and G.P. Dookhran. 1989. Dietary absorption efficiencies and elimination rates of polycyclic aromatic hydrocarbons (PAHs) in rainbow trout (*Salmo gairdneri*). *Environ. Toxicol. Chem.* 8:719-722.

NJDEP. 1993. *Draft Toxic Substances Contamination Assessment of Conditions and Management Recommendations.* New Jersey Department of Environmental Protection, NY/NJ Harbor Estuary Program, Trenton, NJ. August 6.

NJDEP. 1995. Natural Heritage Database Search for Rare, Threatened, or Endangered Species in Passaic River Study Area, Essex and Hudson Counties, NJ. June.

NJMSC. 1987. *The Hudson-Raritan: State of the Estuary*. Summary, Vol. 1, Part 1. Report by The Panel on Water Quality of the Hudson-Raritan Estuary. New Jersey Marine Sciences Consortium. December.

NOAA. 1972. *Final Report The Effects of Waste Disposal in the New York Bight: Section 4. Finfish Studies.* National Oceanic and Atmospheric Administration, Rockville, MD. February.

NOAA. 1981. *Water Quality of the Hudson-Raritan Estuary-Draft.* National Oceanic and Atmospheric Administration, Office of Marine Pollution Assessment, Boulder, CO. NOAA Grant # NA8ORAD00034. November.

NOAA. 1988. *Hudson/Raritan Estuary: Issues, Resources, Status, and Management.* Proceedings of National Oceanic and Atmospheric Administration Estuary-of-the-Month Seminar, February 17, 1987. Seminar Series No. 9. U.S. Department of Commerce. Washington, DC.

NOAA. 1990. *Contaminants in Sediment and Fish Tissue from Estuarine and Coastal Sites of the Northeastern United States: Data Summary for the Baseline Phase of the National Status and Trends Program Benthic Surveillance Project, 1984 - 1986.* National Oceanic and Atmospheric Administration. Technical Memorandum NMFS-F/NEC-79. December.

NOAA. 1991. *National Status and Trends of Marine Environmental Quality Bioeffects Program Intensive Biological Effects Survey Hudson-Raritan Estuary Study Plan.* National Oceanic Atmospheric Administration, Bioeffects Assessment Branch, Coastal Monitoring & Bioeffects Assessments Division, Office of Ocean Resources Conservation and Assessment, Rockville, MD. November.

NOAA. 1993. *Fisheries of the United States 1992.* National Oceanic and Atmospheric Administration, Rockville, MD. May.

NOAA. 1994. *Inventory of Chemical Concentrations in Coastal and Estuarine Sediments.* National Oceanic and Atmospheric Administration, Silver Spring, Maryland. NOAA Technical Memorandum NOS ORCA 76. January.

NOAA. 1995. *Chemical Contaminant Levels in Flesh of Four Species of Recreational Fish from the New York Bight Apex.* National Oceanic and Atmospheric Administration, National Marine Fisheries Service, Highlands, NJ. January 27.

Norstrom, R.J., A.E. McKinnon, and A.S.W. deFreitas. 1976. A bioenergetics-based model for pollutant accumulation by fish: Simulation of PCB and methylmercury residue levels in Ottawa River yellow perch (*Perca flavescens*). *J. Fish. Res. Board Can.* 33:248-267.

NY/NJ HEP. 1993. *Draft: Toxic Substances Contamination Assessment of Conditions and Management Recommendations.* NY/NJ Harbor Estuary Program, Trenton, NJ. August 6.

O'Connor, J.M. and R.J. Huggett. 1988. Aquatic pollution problems, North Atlantic coast, including Chesapeake Bay. *Aquat. Toxicol.* 11:163-190.

O'Connor, J.M. and J.M. Rachlin. 1982. Perspectives on metals in New York Bight organisms: Factors controlling accumulation and body burdens. In: *Ecological Stress and the New York Bight: Science and Management.* G.F. Mayer (eds.). Rockville, MD: National Oceanic and Atmospheric Administration. 655-674.

Odum, E.P. 1972. *Fundamentals of Ecology.* Third edition. Philadelphia, PA: W.B. Saunders Company.

Oliver, B.G. 1984. Uptake of chlorinated organics from anthropogenically contaminated sediments by oligochaete worms. *Can. J. Fish Aquat. Sci.* 41:878-883.

Olsen, C.R., I.L. Larsen, R.H. Brewster, N.H. Cutshall, R.F. Bopp, and H.J. Simpson. 1984. *A Geochemical Assessment of Sedimentation and Contaminant Distributions in the Hudson-Raritan Estuary*. National Oceanic and Atmospheric Administration, Rockville, MD. NOS OMS 2. June.

Padar, F.V. 1990. *The New York - New Jersey Harbor Estuary Study: The Job Begins. Clearwaters* Winter.

Parkerton, T. 1991. *Development of a Generic Bioenergetics-Based Model for Predicting the Bioaccumulation of Persistent Sediment-Associated Contaminants. Appendix 4: Application of a Bioenergetics-Based Bioaccumulation Model for Predicting the Partitioning of 2,3,7,8 Tetrachloro-p-dioxin between Passaic River Biota and Sediment.* NJDEP/EIOSHI Predoctoral Fellowship, Final Report. Rutgers University, Department of the Environmental Science, New Brunswick, NJ and the Manhattan College, Department of Environmental Engineering, Riverdale, NY.

PAS. 1982. Biocommunities Study Passaic Valley Sewerage Commission Combined Sewer Overflow Facilities Plan. Appendix H. In: *Passaic River Sediment Study, Vol. 2.* New Brunswick, NJ: Princeton Aqua Science. July.

Pearce, J.B. 1988. Changing patterns of biological responses to pollution in the New York Bight. In: *Hudson/Raritan Estuary: Issues, Resources, Status, and Management.* Proceedings of National Oceanic and Atmospheric Administration Estuary-of-the-Month Seminar, February 17, 1987. Seminar Series No. 9. U.S. Department of Commerce, Washington, D.C.

Persaud, D. 1993. *Guidelines for the Protection and Management of Aquatic Sediment Quality in Ontario*. Ontario Ministry of the Enviroment, Ontario, Canada.

Pruell, R.J., N.I. Rubinstein, B.K. Taplin, J.A. LiVolsi, and R.D. Bowen. 1993. Accumulation of polychlorinated organic contaminants from sediment by three benthic marine species. *Arch. Environ. Contam. Toxicol.* 24: 290-297.

Purves, W.K. and G.H. Orians. 1983. *Life: The Science of Biology*. Sunderland, MA: Sinauer Associates.

Recher, H.F. and J.A. Recher. 1969. Comparitive foraging efficiency of adult and immature little blue herons (*Florida caerulea*). *Animal Behavior* 17:320-322.

Rod, S.R., R.U. Ayres, and M. Small. 1989. *Reconstruction of Historical Loadings of Heavy Metals and Chlorinated Hydrocarbon Pesticides in the Hudson-Raritan Basin, 1880-1980.* Carnegie Mellon University, Department of Engineering and Public Policy. May.

Rubinstein, N.I., E. Lores, and N.R. Gregory. 1983. Accumulation of PCBs, mercury and cadmium by *Nereis virens, Mercenaria mercenaria* and *Palaemonetes pugio* from contaminated harbor sediments. *Aquatic Toxicol.* 3:249-260.

Rubinstein, N.I., R.J. Pruell, B.K. Taplin, J.A. LiVolsi, and C.B. Norwood. 1990. Bioavailablility of 2,3,7,8-TCDD, 2,3,7,8-TCDF and PCBs to marine benthos from the Passaic River sediments. *Chemosphere* 20(7-9):1097-1102.

Saksena, V.P. and E.B. Joseph. 1972. Dissolved oxygen requirements of newly hatched larvae of the striped blenny (*Chasmodes bosquianus*) and naked goby (*Gobiosoma bosci*) and skilletfish (*Gobiesox strumosus*). *Ches. Sci.* 13:23-28.

Santoro, E.D., N.A. Funicelli, and S.J. Koepp. 1980. Fishes of Newark Bay, N.J. *Bull. Am. Littoral Soc.* 12(2):22.

Setzler, E.M., W.R. Boynton, K.V. Wood, H.H. Zion et al. 1980. *Synopsis of Biological Data on Striped Bass 'Morone saxatilis' (Walbaum).* National Oceanic and Atmospheric Administration, Marine Fisheries Service, Seattle, WA. PB80-225444. June.

Shumway, S.E., T.E. Scott, and J.M. Shick. 1983. The effects of anoxia and hydrogen sulfide on survival, activity, and metabolic rate in the coot clam, *Mulinia lateralis* (Say). *J. Exp. Mar. Biol. Ecol.* 71:135-146.

Sindermann, C.J. and R.L. Swanson. 1979. Historical and regional perspectives. In: *Oxygen Depletion and Associated Benthic Mortalities in New York Bight, 1976*. R.L. Swanson and C.J. Sindermann (eds.). Rockville, MD: National Oceanic and Atmospheric Administration, Professional Paper 11. December.

Solbakken, J.E. and K.H. Palmork. 1981. Metabolism of phenanthrene in various marine animals. *Comp. Biochem. Physiol.* 70C:21-26.

Squires, D.F. 1981. *The Bight of the Big Apple.* The New York Sea Grant Institute of the State University of New York and Cornell University, NY.

Squires, D.F. 1992. Quantifying anthropogenic shoreline modification of the Hudson River and estuary from European contact to modern time. *Coastal Management* 20:343-354.

Squires, D.F. and J.S. Barclay. 1990. *Nearshore Wildlife Habitats and Populations in the New York/New Jersey Harbor Estuary.* Marine Science Institution and Department of Natural Resources Management and Engineering, The University of Connecticut, Storrs, CT. November.

Stacey, P. 1990. Conditions in New York-New Jersey Harbor Estuary. In: *Cleaning Up Our Coastal Waters: An Unfinished Agenda.* A Regional Conference, March 12-14, Manhattan College, Riverdale, NY.

Stainken, D.M. 1984. Organic pollution and the macrobenthos of Raritan Bay. *Environ. Toxicol. Chem.* 3:95-111.

Stickle, W.B., M.A. Kapper, L.L. Liu, E. Gniager, and S.Y. Wang. 1989. Metabolic adaptions of several species of crustaceans and mollusks to hypoxia: Tolerances and microcalorimetric studies. *Biol. Bull.* 177, 303-312.

Suszkowski, D.J., S. Cairns, and D. Heimbach. 1990. Conditions in New York-New Jersey Harbor Estuary. In: *Cleaning Up Our Coastal Waters: An Unfinished Agenda.* A Regional Conference, March 12-14, Manhattan College, Riverdale, NY.

Suter, G.W., II. 1993. *Ecological Risk Assessment.* Ann Arbor: Lewis Publishers.

Targett, T.E. 1979. The effect of temperature and body size on digestive efficiency in *Fundulus heteroclitus (L.). J. Exp. Mar. Biol. Ecol.* 38:179-186.

Theede, H., A. Ponat, K. Hiroki, and C. Schlieper. 1969. Studies on the resistance of marine bottom invertebrates to oxygen-deficiency and hydrogen sulphide. *Mar. Biol.* 2:325-337.

Thomann, R.V. 1989. Bioaccumulation model of organic chemical distribution in aquatic food chains. *Environ. Sci. Technol.* 23(6):699-707.

Thomann, R.V., J.P. Connolly, and T.F. Parkerton. 1992. An equilibrium model of organic chemical accumulation in aquatic food webs with sediment interaction. *Environ. Toxicol. Chem.* 11(5):615-629.

USFWS. 1981. *Fish and Wildlife Resources and Their Supporting Ecosystems; Anadromous Fish Study of the Passaic River Basin, New Jersey.* U.S. Fish and Wildlife Service, Department of the Interior, State College, PA. December.

USFWS. 1985. *Species Profiles: Life Histories and Environmental Requirements of Coastal Fishes and Invertebrates (Mid-Atlantic): Mummichog and Striped Killifish.* U.S. Fish and Wildlife Service, Department of the Interior, Washington, D.C. Biological Report 82(11.40). June.

USFWS. 1989. *Species Profiles: Life Histories and Environmental Requirements of Coastal Fishes and Invertebrates (Mid-Atlantic): Blue Crab.* U.S. Fish and Wildlife Service, Department of the Interior, Washington, D.C. Biological Report 82(11.100). March.

Valiela, I., J.E. Wright, J.M. Teal, and S.B. Volkmann. 1977. Growth, production and energy transformations in the salt-marsh killifish *Fundulus heteroclitus. Marine Biology* 40:135-144.

Varanasi, U. and D.J. Gmur. 1981. Hydrocarbons and metabolites in English sole (*Parophrys vetulus*) exposed simultaneously to [3H]benzo[a]pyrene and [14C]naphthalene in oil-contaminated sediment. *Aquat. Toxicol.* 1:49-67.

Varanasi, U. and J.E. Stein. 1991. Disposition of xenobiotic chemicals and metabolites in marine organisms. *Environ. Health. Perspect.* 90:93-100.

Varanasi, U., W.L. Relchert, J.E. Stein, D.W. Brown, and H.R. Sanborn. 1985. Bioavailability and biotransformation of aromatic hydrocarbons in benthic organisms exposed to sediment from an urban estuary. *Environ. Sci. Technol.* 836-841.

WADOE. 1991. *Sediment Management Standards; Chapter 173-204 WAC.* Washington Department of Ecology, Olympia, WA. April.

Wallace, R. and T. Wallace. 1983. *A Development Plan for the Newark Passaic Riverfront Downtown Newark, New Jersey.* Final Report. Architects, Landscape Architects, Urban and Ecological Planners, Philadelphia, PA. February 21.

Weinstein, M.P., S.L. Weiss, and M.F. Walters. 1980. Multiple determinants of community structure in shallow marsh habitats, Cape Fear River Estuary, North Carolina, USA. *Mar. Biol.* 58:227-243.

Weisberg, S.B., R. Whalen, and V.A. Lotrich. 1981. Tidal and diurnal influence on food consumption of a salt marsh killifish *Fundulus heteroclitus. Marine Biology* 61:243-246.

Wenning, R.J. and G.A. Erickson. 1994. Interpretation and analysis of complex environmental data using chemometric methods. *Trends Analy. Chem.* 13(10):446-458.

West, D.L. and A.H. Williams 1986. Predation by *Callinectes sapidus* (Rathbum) within *Spartina alterniflora* (Loisel) marshes. *J. Exp. Mar. Biol. Ecol.* 100:75-95.

Woodhead, P.M.J. 1991. *Inventory and Characterization of Habitat and Fish Resources and Assessment of Information on Toxic Effects in the New York - New Jersey Harbor Estuary: A Report to the New York-New Jersey Harbor Estuary Program Concerning Work in Tasks 3.2, 5.1 and 5.3.* Marine Sciences Research Center, State University of New York, Stony Brook, New York. May.

Wyman, K.D. and H.B. O'Connors. 1980. Implications of short-term PCB uptake by small estuarine copepods (*Genus Acartia*) from PCB-contaminated water, inorganic sediments and phytoplankton. *Estuar. Coast. Mar. Sci.* 11:121-131.

Zdepski, J.M. 1992. *Industrial Development, Urban Land-Use Practices, and Resulting Groundwater Contamination, Newark, NJ.* NGWA Focus Eastern Conference, Boston, MA. October 13-15.

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